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ORIGINAL ARTICLES

CHOLANGIOGRAPHIE PER-OPERATOIRE*

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INVENTÉE en 1932, par Marrizi, la cholangiographie per-opératoire ne s'est imposée que plusieurs années plus tard dans les milieux chirurgicaux. L'abondance des publications récentes sur le sujet, témoigne de plus en plus de l'intérêt que l'on y porte actuellement.

Dans notre milieu, cette technique est pratiquée depuis plus de cinq ans. Nous avons pensé qu'il serait intéressant d'étudier nos résultats pour les comparer à ceux de la littérature médicale actuelle.

Les buts de la cholangiographie peuvent se résumer comme suit: 1.—éviter les cholédocotomies inutiles. 2.—découvrir des pathologies insoupçonnées. 3.–découvrir des calculs insoupçonnées. 4.—diminuer le nombre de calculs résiduels.

I. MATÉRIEL

Nous avons réuni, sur une période de cinq ans (1956-1960) 1764 dossiers comprenant 1740 cholécystectomies et 24 cholédocotomies chez des malades antérieurement cholécystectomisés. De ces dossiers, nous avons pu extraire 145 cholangiographies per-opératoires effectuées avec un tube de polythène par voie cystique, 85 cholangiographies per-opératoires par tube en T, faisant suite à des cholédoctomies, et finalement 94 cholédoctomies sans cholangiographie per-opératoire.

II. TECHNIQUE DE LA CHOLANGIOGRAPHIE PER-OPÉRATOIRE

La technique que nous suivons, se superpose à celle décrite par Nienhuis.[15] Un matelas spécial est installé sur la table d'opération. Il permet de glisser facilement la cassette contenant la pellicule radiographique, sous le patient. Un appareil portatif de rayons x est avancé au-dessus du malade au moment voulu. Dès que le carrefour hépatique est disséqué, le cystique est ouvert et canulé avec un tube de polythène. Une première radiographie est prise après injection de 2 c.c. de "diodrast" et une deuxième après 10 c.c. La vésicule est enlevée pendant qu'on procède au développement des radiographies. Le temps additionnel nécessité par cette manœuvre est d'environ 10 minutes.

III. ETUDE DES DOSSIERS

Notre étude se divise en trois parties: (a) Cholangiographie par voie cystique à l'aide d'un tube de polythène; (b) cholangiographie par tube en T après cholédocotomie; (c) analyse des cholédocotomies où la cholangiographie n'a pas été utilisée.

(a) Cholangiographie par tube de polythène (tableau I). (145 cas)

TABLEAU I.—CHOLANGIOGRAPHIE PAR TUBE DE POLYTHÈNE (145 CAS)

Douteuse	3	
Erreur technique	5	
Positive	23	
Néo du pancréas		1
Sténose de l'Oddi		2
Calcul trouvé		13
Calcul non trouvé		7
Faussement positifs		5
Négative	114	
Pas de cholédocotomie		107
Cholédocotomie faite		7
Faussement négatif		1

Erreurs: 6 sur 145 cas soit: 4.1%

Cinq erreurs techniques n'ont pas permis d'interprétation. La cholangiographie s'est averée douteuse dans trois cas. On a dû faire une cholédocotomie dans un seul de ces cas et elle s'est avérée négative. Dans 23 cas, la cholangiographie s'est montrée positive. Dans un cas, il s'agissait d'un néo du pancréas qui a nécessité une cholédoco-jéjunostomie. Dans deux cas, on a dû pratiquer une sphinctérotomie pour sténose du sphincter d'Oddi. Dans sept cas, les calculs ne furent pas découverts, bien que présents dans deux de ces cas, et il fallut réopérer. Les cinq autres cas étaient

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TABLEAU II.—Cholangiographie sur tube en T après cholédocotomie (85 cas)

Douteuse	2		
Erreur technique	1		
Calcul trouvé	48 (55%)		
—Cholangiographie positive	10		
(a) Réexploration	8	Calcul trouvé	5
		Calcul non trouvé	2 faux +
		Sphinctérotomie	1
(b) Pas de réexploration	2		
Cholangiographie post-opératoire		Négative	1 faux +
		Positive	1
—Cholangiographie négative	38		
Cholangiographie post-opératoire		Négative	30
		Positive	2 (dont 1 faux−)
Pas de cholangiographie post-opératoire			6
Pas de calcul trouvé 34 (40%)			
—Cholangiographie positive	3	Néo des voies biliaires	2
		Calcul	1
—Cholangiographie négative	31		
Cholangiographie post-opératoire		Négative	20
		Positive	2 (faux−)
Pas de cholangiographie post-opératoire			9

Erreurs: 6 sur 85 cas soit: 7%.

faussement positifs et la cholangiographie post-opératoire resta négative. On a trouvé des calculs dans 13 cas.

La cholangiographie s'est avérée négative dans 114 cas. Dans sept cas, on a tout de même fait une cholédocotomie: une seule a permis l'exérèse d'un calcul de 3 mm. Dans les 107 autres cas, on n'a pas fait de cholédocotomie: 29 de ceux-ci, étaient ictèriques ou avaient un passé d'ictère; 12 avaient un cholédoque dilaté, de plus de 8 mm. de diamètre. Les calculs vésiculaires étaient petits dans 51 cas. Les 14 autres cas ont été faits de routine.

Nous relevons donc ici cinq cas faussement positifs (c'est moins grave d'avoir des faux positifs que des faux négatifs) et un cas faussement négatif, soit six erreurs sur 145 cas ce qui nous donne un taux de 4.1%. Ces erreurs seront étudiées plus loin.

(b) Cholangiographie per-opératoire sur tube en T (tableau II).

Nous avons relevé 85 cas de cholédocotomies où après celles-ci on a fait une cholangiographie per-opératoire. Cette dernière s'avéra douteuse dans deux cas. A la vérification, on trouva des calculs dans les deux cas. Un cas n'a pas permis d'interprétation: il s'agissait d'un tube de Cattel passant dans le duodénum, et l'opacification des voies biliaires était insuffisante. Des calculs avaient été trouvés à la cholédocotomie dans 48 cas (55%). La cholangiographie par tube en T fut positive dans 10 cas. Huit furent réexplorés: un dut avoir une sphinctérotomie, cinq présentaient des calculs résiduels, deux ne présentaient aucune pathologie. Deux cas ne furent pas réexplorés: la cholangiographie post-opératoire fut négative chez l'un et positive chez l'autre. Ce dernier fut réopéré, mais il fut impossible d'extraire le calcul enclavé dans la branche hépatique gauche.

La cholangiographie sur tube en T fut négative dans 38 des 48 cas où des calculs avaient été enlevés. Une cholangiographie post-opératoire s'avéra négative dans 30 cas et positive dans deux: un cas présentait une obstruction externe du tube en T par des concretions biliaires et l'autre un calcul dans une branche hépatique.

Dans 34 cas de cholédocotomies, aucun calcul n'a pu être trouvé (39%). La cholangiographie per-opératoire a été positive dans trois cas: deux de ceux-ci correspondaient à des néoplasies des voies biliaires, l'autre à un calcul résiduel. La cholangiographie fut négative dans 31 cas: deux étaient faussement négatifs et durent être opérés pour calculs résiduels. On peut relever ici, six erreurs sur 85 cas soit 7%. Notons aussi, que 39% des cholédocotomies étaient inutiles.

(c) Cholédoctomie sans cholangiographie per-opératoire (tableau III).

Quatre-vingt-quatorze cas ont été étudiés. Des calculs furent trouvés 51 fois. La cho-

TABLEAU III.—CHOLÉDOCOTOMIE SANS CHOLANGIOGRAPHIE PER-OPÉRATOIRE (94 CAS)

Calcul trouvé: 51 cas (54.3%)

—Cholangiographie post-opératoire.... 35

Positive { Néo du pancréas..... 1 / Calcul résiduel..... 4 (11.4%)
Négative..... 30

—Pas de cholangiographie post-opératoire 16

Pas de calcul trouvé: 43 cas (45.7%)

—Cholangiographie post-opératoire.... 35

Positive — Néo de l'ampoule..... 1
Négative..... 34

—Pas de cholangiographie post-opératoire 8 dont 5 décès (11.6%)

langiographie post-opératoire (35 cas), s'est avérée positive dans cinq cas (un néo du pancréas, quatre calculs résiduels). Il y a donc des calculs résiduels dans 11.4% (4/35) des cholédocotomies où l'on a trouvé des calculs. On n'a pas fait de cholangiographies dans 16 cas: quatre de ceux-ci étaient décédés.

La cholédocotomie n'a pas démontré de calcul dans 43 cas (45.7%). Une seule cholangiographie post-opératoire fut positive: il s'agissait d'un cas de néoplasie de l'ampoule de Vater vérifié à l'intervention. On n'a pas fait de cholangiographies dans huit cas. Cinq de ceux-ci étaient décédés après l'opération représentant 11.6% des cholédocotomies inutiles.

IV. ANALYSE DE NOS RÉSULTATS

(a) Cholangiographies à l'aide d'un tube de polythène

1. *Diminution du nombre des cholédocotomies inutiles.*—L'opinion varie chez les divers auteurs de savoir, si la cholédoctomie augmente la mortalité. Colcock[7] estime que non. Par contre plusieurs auteurs[6, 13, 15, 25] pensent que la mortalité passe de 1% à 3% ou 5%, quand la cholédocotomie est ajoutée à la cholécystectomie. Mais ceci semble dû plutôt à la présence de calculs dans le cholédoque qu'à la cholédocotomie elle-même. La cholédocotomie blanche serait inoffensive. Cependant, sur les 94 cholédoctomies sans cholangiographies, nous trouvons une mortalité totale de 9.2% (neuf décès sur 94) se répartissant ainsi: quatre décès chez les 51 cas porteurs de calculs (7.8%) et cinq chez les 43 cas de cholédocotomies inutiles (11.6%). On ne peut donc pas considérer la cholédocotomie comme entièrement inoffensive.

Par contre, il est généralement admis que la cholédocotomie augmente le temps d'hospitalisation.

Or, sans cholangiographie, on est amené à faire énormément de cholédocotomies inutiles. Ykelenstam[2] rapporte 50 explorations dans 100 cholécystectomies, dont 23 inutiles. Pour Sachs,[4] grâce à la cholangiographie, les explorations inutiles diminuent de 45% ou 50%, à 4% ou 5%. Mixter[6] rapporte un taux de 45% de cholédocotomies inutiles sur 297 explorations. Gardner *et al.*[8] rapportent eux aussi 49.5% de cholédocotomies blanches sur 104 explorations. Isaacs et Daves[11] sur 150 explorations rapportent 62% d'explorations vaines. Colcock[7] explorant un tiers des cas, trouve des calculs dans moins d'un tiers des explorations. Bartlett et Waddell[23] rapportent 43% d'explorations sur 2,243 cas: 16% seulement révélèrent des calculs. Nos chiffres concordent avec ces derniers puisque sur 94 cholédocotomies, 43 étaient inutiles, soit 45.7%.

Il est évident que la cholangiographie diminue le nombre de cholédocotomies blanches. Dans nos 145 cas de cholangiographies par le cystique, 32 explorations (17%) furent faites dont cinq inutilement (3.4%). Ykelenstam[2] rapporte 19 explorations sur 100 cas de cholangiographies dont quatre inutiles. Ceci correspond aux résultats de Swedberg,[1] Sachs,[4] Mixter, Hermanson et Segel,[6] Isaacs et Daves,[11] Maingot,[13] et Nienhuis.[15] On peut donc admettre que le nombre des cholédocotomies inutiles diminue de 40% ou 50% à 4% ou 5%.

2. *Découverte de calculs insoupçonnés.* (Fig. 1).—La cholangiographie par le cystique permet souvent de découvrir des calculs insoupçonnés dans le cholédoque. Ainsi Swedberg[1] rapporte que sur 700 cas de cholécystectomies, dans 16.05%, il a repéré des calculs du cholédoque en utilisant la cholangiographie. Par contre sur 768 cholécystectomies sans cholangiographie, il en a trouvé dans seulement 8.47% des cas. Dans 22 cas (3.1%) où la

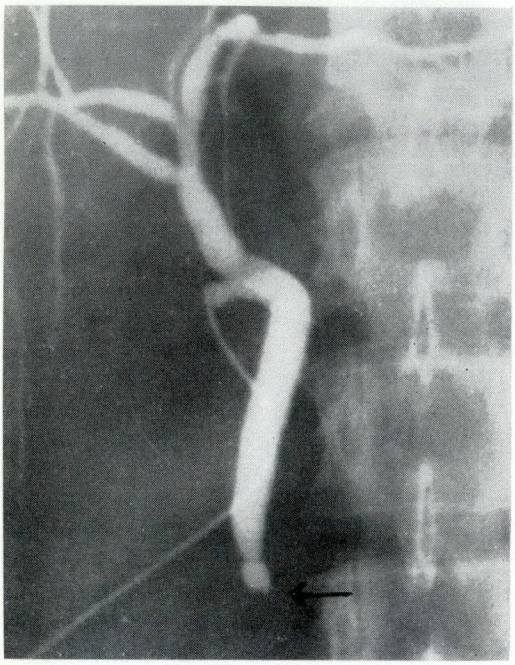

Fig. 1.—Cholangiographie sur tube de polythène (Mme. J.L., 45 ans). Très gros calculs vésiculaires. Il n'y avait aucune indication de cholédocotomie. La cholangiographie de routine a montré la présence d'un petit calcul de 3 mm. de diamètre bloquant complètement l'ampoule de Vater, causant une fibrose du sphincter. Une sphinctérotomie dût être pratiquée.

Fig. 2.—Anomalie congénitale (Mme. J.T. 56 ans). Ce cas récent, n'est pas inclu dans notre statistique. La cholangiographie a montré un double abouchement du cholédoque dans le duodénum. Ceci fut vérifié par duodénotomie.

cholangiographie fut faite, les calculs seraient passé inaperçus. Gardner *et al.*[8] rapportent un pourcentage de 2.2% de calculs découverts fortuitement sur 136 cholécystectomies. Isaacs et Daves ont trouvé des calculs dans 7 sur 87 (8%) cas de cholangiographies de routine. Sur 171 cholangiographies, Nienhuis[15] rapporte sept cas de calculs non suspectés, soit un pourcentage de 4.1%. Cette même valeur est de 10% chez Mehn,[18] et de 26.4% chez Ferris et Weber[16] (185 cholangiographies). Personnellement nous relevons neuf cas sur 145 cholangiographies soit 6%.

3. *Découverte d'affections insoupçonnées.*—La cholangiographie peut permettre souvent de mettre en évidence des pathologies ou des anomalies insoupçonnées. Ainsi, Isaacs et Daves[11] rapportent trois cas d'anomalies congénitales sur 87 cholangiographies de routine (3.4%). Nienhuis[15] rapporte une tumeur des voies biliaires (.6%), deux fibroses du sphincter d'Oddi (1.2%), deux cas d'anomalies congénitales (1.2%) sur 171 cholangiographies. Rosenqvist[25] et Le Quesne concèdent aussi cette valeur à la cholangiographie.[13] Nous-même avons trouvé deux cas de sténose du sphincter d'Oddi (1.3%) dans 145 cholangiographies cystiques. Tout récemment un cas de bifurcation en Y du cholédoque fut mis en évidence chez une patiente (Fig. 2), ainsi qu'un cas de néoplasie des voies biliaires (Fig. 3).

4. *Les calculs intra-pancréatiques et la dilatation du cholédoque.*—La littérature reste muette en ce qui concerne la cholangiographie et la localisation des calculs. Nous croyons que les calculs intra-pancréatiques sont très difficilement palpables sauf s'ils sont assez volumineux.

Aussi chez 13 malades où la cholangiographie par le cystique, a permis de mettre en évidence des calculs, les calculs n'ont

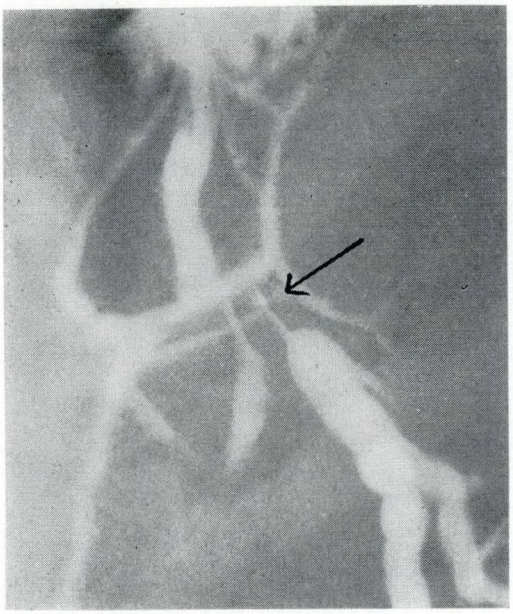

Fig. 3.—Néoplasie de la branche hépatique droite (Mlle S.C., 45 ans). Opérée pour ictère. La cholédocotomie est normale. Grâce à la cholangiographie on a pu déceler un néo de la branche hépatique droite, obstruant la bifurcation.

été palpés que trois fois. Dans 10 cas, les calculs étaient situés dans le cholédoque intra-pancréatique ou dans l'ampoule de Vater. Dans neuf de ceux-ci (72%) le cholédoque n'était pas dilaté. Les calculs intra-pancréatiques sont donc difficiles à palper et l'absence de dilatation du cholédoque ne signifie pas qu'il n'y a pas de calculs.

(b) Cholangiographies per-opératoires par tube en T

1. *Calculs résiduels et cholangiographies* (Fig. 4).—Il est difficile d'évaluer statistiquement le pourcentage de calculs résiduels après cholédocotomie et exploration. Maingot[13] croit que l'incidence est de l'ordre de 6% à 10%. Sherman et Stabins[3] ont publié trois statistiques où l'incidence est de 2.5% à 20%. Johnston, Waugh et Good[5] donnent une valeur de 8% (sur 175 cholédocotomies); Isaacs et Daves[11] 10.5% (sur 57 cholédocotomies); Buxton et Burk[19] 8.3%; Smith *et al.*,[20] 10.7%; Thomson[21] 11.6% et Paine et Firme[22] 27% (sur 206 explorations).

Or la cholangiographie per-opératoire par tube en T permet d'abaisser ces taux

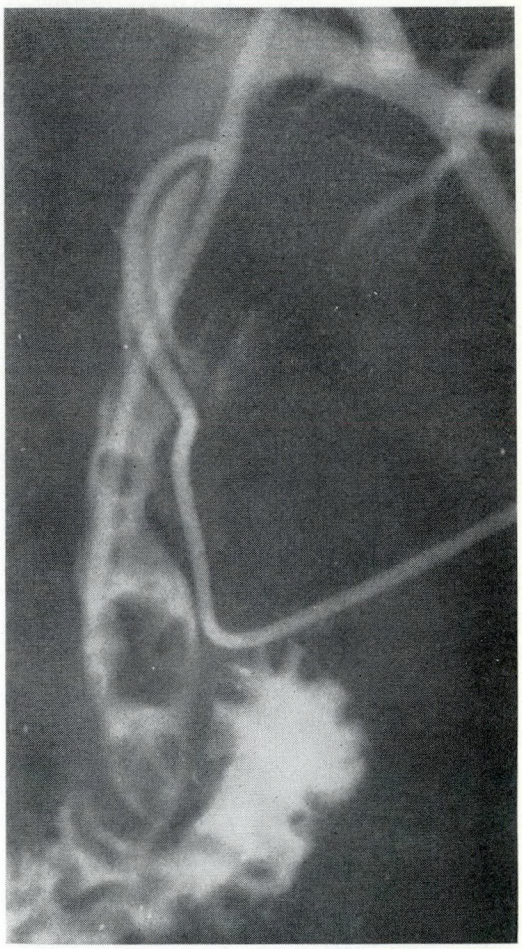

Fig. 4.—Cholangiographie per-opératoire après cholédocotomie (M. R.R. 45 ans). Plus de 10 calculs avaient été extraits lors de la cholédocotomie. On voit l'avantage de la cholangiographie. Il restait encore quatre calculs.

de façon très importante. Sachs[4] estime avec justesse que ceux-ci passent de 16% ou 25% à 4%. Mixter (cité par Sherman et Stabins[3]) rapporte 72 cas sans calculs résiduels. Nienhuis[15] a 4.3% de calculs résiduels (39 explorations); il a pu les faire disparaître par injection d'éther dans le tube en T. Il cite aussi Mixter *et al.* qui ont un taux de 4.6% et Hicken *et al.* 3.5%. Quant à nous, la cholangiographie a permis d'identifier des calculs résiduels dans 14% (48 malades) des explorations positives.

2. *Cholangiographie par tube en T et syndromes divers.*—La cholangiographie par tube en T permet aussi de découvrir des maladies insoupçonnées lors de l'ex-

ploration. Dans 8.8% des cholédocotomies blanches (34 malades) on a pu reconnaître deux néoplasies des voies biliaires et un cas de calcul résiduel.

3. *Cholangiographie post-opératoire par tube en T.*—Il faut toujours effectuer une cholangiographie avant d'enlever un tube en T. La technique radiologique est plus précise que lors de l'opération. On pourra ainsi abaisser le taux d'erreur inhérent à la cholangiographie per-opératoire. Ainsi sur 70 cas où la cholangiographie fut faite post-opératoirement sur le tube en T, on a pu découvrir quatre cas de calculs résiduels.

V. Analyse des Erreurs de la Cholangiographie

(*a*) *Fréquence des cas faussement positifs ou faussement négatifs*

Isaacs et Daves[11] donnent un taux d'erreur de 4.1%, Nienhuis[15] de 3.4%, Hicken *et al.* (cités par Nienhuis[15]) de 3.5%, et Mixter de 4.6%. Or notre taux d'erreur est plus élevé: il s'élève à 4.1% (six cas) dans les 145 cholangiographies par le cystique et à 7% (six cas) dans les 85 cholangiographies per-opératoires sur tube en T.

(*b*) *Analyse des cas précédents*

Sur les 145 cholangiographies par le cystique, on a eu six erreurs. Dans un cas on n'a pas pu retrouver les clichés et on trouve de l'air dans trois cas. Chez un malade un calcul a probablement été poussé dans le duodénum. Dans un autre cas, le calcul aurait du être soupçonné sur la radiographie. Dans quatre de ces cas, les erreurs auraient pu être facilement évitées.

Sur les 85 cholangiographies sur tube en T, on a eu six erreurs. Pour un cas les films n'ont pas pu être retrouvés, et dans un autre cas, on trouve une bulle d'air. Chez un autre malade, les clichés sont mal centrés, trop injectés. Dans trois de ces cas, le calcul est bien visible mais les rapports sont négatifs: il s'agit d'erreurs radiologiques pures. Chez un patient le calcul aurait dû être soupçonné radiologiquement: une branche hépatique est amputée et mesure 1 cm. de diamètre. Ici donc, on se trouve devant trois erreurs radiologiques grossières qui auraient facilement pu être évitées, ce qui baisse le taux d'erreur à 3.4%.

(*c*) *Sources d'erreurs*

Les erreurs dans les cholangiographies peuvent être causées par: (1) une mauvaise position du malade; (2) une cholangiographie faite à l'imprévu; (3) l'absence de radiologiste sur les lieux; (4) l'heure tardive de l'opération—manque de personnel; (5) l'appareil à rayons x portatif; (6) l'injection de bulles d'air; (7) une trop grande quantité de liquide lors de la première injection (2 c.c.); (8) les mouvements respiratoires du malade au moment de l'exposition.

(*d*) *Autres causes*

D'autres explications à notre taux d'erreur peuvent être données. Nos statistiques englobent les résultats de 14 chirurgiens auxquels viennent se joindre chaque année trois ou quatre résidents. Aussi bon nombre de chirurgiens ne sont pas convaincus de l'utilité de la cholangiographie. D'autres en font depuis peu seulement. Or il est prouvé que le pourcentage d'erreurs diminue avec l'expérience du chirurgien. Dans ces conditions le peu d'erreurs observé dans nos cas est étonnant.

VI. Conclusion

La cholangiographie per-opératoire devient de plus en plus répandue. Maingot[13] dans la récente édition de "Abdominal Operation" lui consacre une place importante: et admet en faire de plus en plus. Rosenqvist[25] dit que cette technique fut introduite en Suède en 1936, soit quatre ans seulement après que Marrizi l'eut proposée. Depuis 20 ans, elle est couramment pratiquée dans les pays scandinaves. En Amérique la méthode est de plus en plus utilisée. Nous n'avons trouvé que deux articles[5-7] qui tendent à la condamner. Une nouvelle technique apparaît: la cholédocoscopie[10] qui peut-être la remplacera.

On peut donc conclure:

1o — La cholangiographie par le cystique sur tube de polythène: (a) permet de

diminuer le nombre de cholédocotomies inutiles de 45% ou 50% à 3% ou 5%; (b) permet de mettre en évidence des calculs insoupçonnées dans 4% à 8% des cas; (c) permet de découvrir des maladies non soupçonnées: anomalies congénitales —sténose du sphincter—néo des voies biliaires.

2o — Qu'il est impossible de se fier à la palpation pour repérer les calculs du cholédoque intra-pancréatique ou de l'ampoule de Vater.

3o — La cholangiographie sur tube en T après exploration est nécessaire car: (a) 8% à 15% des explorations doivent être refaites après cholangiographie; (b) le pourcentage de calculs résiduels tombe de 10% ou 20% à 3% ou 5% si on fait la cholangiographie; (c) elle peut permettre de découvrir une affection expliquant parfois l'absence de calcul (néoplasie-rétrécissement).

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Summary

The authors have reviewed the cholangiograms performed during operations on the biliary tract over a period of five years in the Notre-Dame Hospital, Montreal. The results of 145 cholangiograms performed with a polyethylene tube inserted into the cystic duct and of 85 cholangiograms performed through the T-tube after choledocotomy are compared with those of 94 choledochotomies in which no cholangiography was carried out during the operation. The results of this study are compared with the findings reported by others in the literature. Sources of errors in cholangiography are analyzed.

It is concluded that: 1. Cholangiography through the cystic duct with a polyethylene tube leads to (a) a decrease in the number of useless choledochotomies from 45-50% to 3-5%; (b) visualization of unsuspected calculi in 4 to 8% of cases, and (c) the discovery of unsuspected pathology: congenital anomalies, stenosis of the sphincter and neoplasms of the biliary tract. 2. It is unwise to rely upon palpation to identify calculi of the intrapancreatic portion of the common duct or of the ampulla. 3. Preoperative T-tube cholangiography, after exploration of the biliary tract, is imperative because (a) 8 to 15% of the explorations must be repeated after cholangiography; (b) residual stones can thereby be reduced from 10-20% to 3-5%, and (c) underlying pathology, explaining the failure to find calculi, is occasionally discovered (neoplasms and stenosis).

EWING'S TUMOUR: A REVIEW OF 33 CASES

RONALD J. BAIRD, M.D., F.R.C.S.[C]* and VERNON W. KRAUSE, M.D.,† *Toronto*

In 1921, James Ewing[1] described the pathological features of a tumour which appeared in the bones of young people and was characterized by a distinctive radiological appearance, a rapid initial response to radiotherapy and a fatal outcome. This tumour is still of great interest, not only because of its lethal nature, but also because of the controversy over its cellular origin — that is, whether it arises from the endothelium of the marrow spaces or from the connective tissue framework of the marrow.[2, 3]

The purpose of this article is to review and analyze the clinical course and the pathological features of 33 cases of Ewing's sarcoma diagnosed and treated at The Hospital for Sick Children, Toronto. The diagnosis was recorded for the first time in this hospital in 1928; the most recent case reviewed was seen in June 1960.

A. Clinical Picture

There were 20 girls (60%) and 13 boys in this series. This slightly increased incidence in females is unusual in Ewing's tumour; other large series have reported a male preponderance which varied from 60% to 75%.[4-6]

Because of the age limit governing admission to The Hospital for Sick Children, all of the patients were under 16 years of age; the youngest was 13 months old. Eighty per cent of the children were between the ages of seven and 14 years when the diagnosis was made (Fig. 1). In the largest reported series of patients with this tumour, 165 patients seen at the Mayo Clinic up to 1960, the peak incidence was in the second decade of life and almost 90% were under 30 years of age.[6]

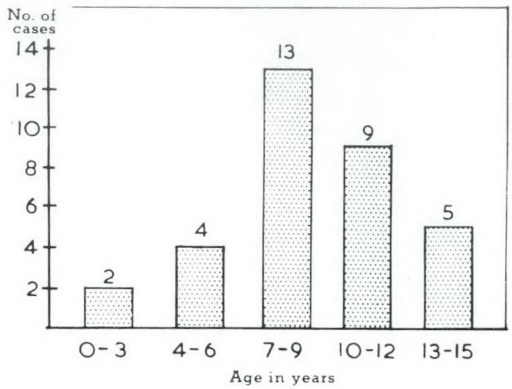

Fig. 1.—The age at diagnosis of the 33 patients with Ewing's tumour.

Symptoms and Signs

There was no symptom or sign which specifically indicated the presence of this particular tumour. The symptoms which were present at the time of diagnosis are presented in Table I. Local swelling, pain, general malaise and a low-grade fever were all common complaints. Pain at the site of the tumour was the first symptom in over two-thirds of the children. A pathological fracture was present in only one child. In five of the patients the symptoms seemed to be related to injuries which had occurred from one to six months before the onset of symptoms.

In only six of the children was the diagnosis made within one month of the onset of the symptoms; in 20, symptoms had been present from two to six months; and in seven, for over six months. It is of interest that one child, who complained of vague aches in the arm for over two years before diagnosis of Ewing's tumour of the radius, is one of the long-term survivors.

In the course of the initial hospital examination, a mass was palpable in nearly every child (29/33). The examining doctor stated that the area was warm to touch in six children, but he rarely noted enlarged superficial veins or local redness. In one child the presence of the tumour was announced by a painless progressive paraplegia.

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TABLE I.—Symptoms Present at Time of Diagnosis in 33 Children with Ewing's Sarcoma

Pain	31
Swelling	26
Fever	8
Pathological fracture	1
Other	1

Investigation

Although pallor and anemia are stated to be common findings in this condition, the hemoglobin level was below 12 g. % in only one-half of the children. The leukocyte count ranged from 4600 to 26,000 per c.mm. and was usually within normal limits. The Westergren sedimentation rate varied from 8 mm. to 38 mm. per hour.

The basic features of the radiological appearance of this tumour — a mottled lysis, sclerosis, cystic loculation and periosteal reaction—have been well described elsewhere.[4] The classical "onion-skin" appearance was seen only occasionally. Although the radiological appearance and clinical picture may allow a highly presumptive diagnosis of this tumour, it is essential that an adequate biopsy be obtained before commencing treatment. Biopsies were performed in 32 of the 33 patients reported here; the diagnosis in the remaining child was confirmed at postmortem examination.

Location

The location of the tumours is shown in Table II. The innominate bone, the humerus and the tibia were the most common sites; the ribs, the vertebrae and the skull were rarely involved.

TABLE II.—Location of the Primary Tumour in 33 Children with Ewing's Sarcoma

Trunk:		
Skull	1	18%
Vertebrae	1	
Ribs	4	
Upper extremity:		
Scapula, clavicle	2	33%
Humerus	6	
Radius	1	
Hand	2	
Lower extremity:		
Innominate	7	49%
Femur	3	
Tibia	5	
Foot	1	

B. Pathology

The following criteria for classifying a neoplasm as Ewing's tumour have been used at The Hospital for Sick Children: (a) The tumour is unquestionably primary in bone. (b) The tumour cell is undifferentiated. (c) Although reactive bone formation may be found, bone or related tissues are not produced by the tumour cells.

In the gross, a large portion of the tumour may be necrotic and appear "mushy", soft, greyish-yellow and hemorrhagic. The viable portions are found in the marrow and are glistening, silvery-grey and nodular. Whenever possible, viable tissue should be secured for biopsy because it affords the best chance for diagnosis. In one of our patients, the diagnosis was missed on the initial biopsy when only thickened cortical tissue was submitted.

Microscopically, these tumours are composed of sheets of cells which have ill-defined borders, meagre cytoplasm and fairly large, prominent, uniform, round or oval nuclei with scattered chromatin.[8] However, the necrosis and degeneration, so frequently present, may alter the histological appearance and lead to the formation of the polyhedral cells with pale cytoplasm and well-defined borders, originally described by Ewing.[1]

Willis[7] has questioned the very existence of this neoplasm. He states, "Ewing's tumour is not a pathological entity; it is merely a (rather ill-defined) syndrome of a non-osteogenic, round-celled, radiosensitive tumour in a bone, usually a long bone, usually in a young subject, and usually causing diffuse elevation of the periosteum." This syndrome, he says, can be caused by several different kinds of tumours, such as metastatic neuroblastoma in children and adolescents, metastatic carcinoma in adults and, rarely, primary reticulum-cell sarcoma.

Although it is not the purpose of this paper to enter into a defence of the pathological entity of Ewing's tumour, it is our view that the following findings justify its consideration as a pathological as well as a clinical entity: (a) Eight autopsies were performed in this series. None showed evi-

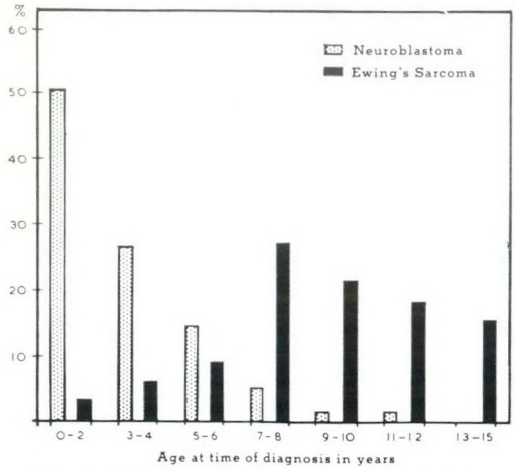

Fig. 2.—The definite difference in age incidence of patients with Ewing's tumour (33) and those with neuroblastoma (109).

dence of neuroblastoma. (b) The age incidence of neuroblastoma is quite different from that of Ewing's tumour (Fig. 2). As the incidence of our proved cases of neuroblastoma falls from its peak in infancy, the incidence of Ewing's tumour rises to reach a peak in late childhood.

The differentiation of Ewing's tumour from reticulum-cell sarcoma of bone is difficult. Nevertheless, reticulum-cell sarcomas occur in older patients, grow less rapidly and are composed of cells which are more pleomorphic. In the Tumour Registry of The Hospital for Sick Children, Toronto, where the patients are all under the age of 16 years, no cases of primary reticulum-cell sarcoma of bone have ever been recorded.

Other tumours in bone which may sometimes simulate Ewing's tumour are lymphosarcoma, metastatic carcinoma and eosinophilic granuloma. When the histological material is inadequate for precise diagnosis, an evaluation of the overall clinical picture and laboratory findings (blood and bone marrow smears, radiographs and postmortem features) should furnish a correct diagnosis in the vast majority of such lesions.

C. Treatment and Results

The methods employed at this hospital in treating patients with Ewing's tumour were local and radical surgery, radiation and chemotherapy. In several children more than one method was used (Table III). The follow-up study to August 1961 is complete in 32 of the 33 children. One child, treated in 1947, was lost to follow-up in 1950. The results of treatment are presented in Table IV.

TABLE III.—Method of Therapy in 33 Cases of Ewing's Sarcoma

Radiation alone	17
Operation alone	5
Operation plus radiation	8
Perfusion plus radiation	1
No treatment	2

TABLE IV.—Results of Treatment in 33 Children with Ewing's Sarcoma

	Number of patients	*%*
Survival for over 5 years	3	9
Survival for 3 to 5 years	2	6
Survival for 1 to 3 years	5	15
Survival for less than 1 year	21	64
Treated within past year and still alive	2	6
	33	100

Five-Year Survivals—3/33

The lethal nature of the tumour is indicated by the five-year survival of only three of the 33 patients (9%). In 1935, a 10-year-old girl with a tumour of the ilium was treated by radiation. In 1940, a 6-year-old boy with a tumour in the shaft of the radius underwent a disarticulation at the elbow. In 1954, a 7-year-old girl with a tumour in the shaft of the fourth metacarpal was treated by amputation of the third, fourth and fifth metacarpals and fingers. All three patients are alive and well and have no evidence of recurrence.

Three-Year Survivals—2/33

One child died in the fourth year after a disarticulation at the knee for a lesion in the tibia. A second child, with a tumour in the humerus, was treated by curettage; she was well at three years and was then lost to follow-up study.

One-Year Survivals—5/33

Four children died between the first and third years after treatment. Radiation was employed twice (femur and metacarpal)

and operation followed by radiation twice (tibia and scapula). The fifth patient, a 13-year-old boy with a Ewing's tumour in the tibia, was treated by perfusion with nitrogen mustard. The leg was isolated by the technique described by Creech *et al.*,[9] and the heart-lung pump attached to the superficial femoral artery and vein. The nitrogen mustard was given in a dosage of 0.6 mg./kg. of body weight; this equalled 3.0 mg./kg. of perfused tissue. The tumour decreased in size and the radiographical picture remained static for six months. Local progression was then noted and he was referred for radiation. Nevertheless, widespread secondaries appeared and despite adjunctive systemic chemotherapy he died from the tumour 18 months after the first symptom appeared.

Survival Less than One Year—21/33

Two-thirds of the children were dead within one year after diagnosis. Treatment consisted of radiation alone, 12 patients; radiation plus operation, five patients; operation alone, two patients; and no specific treatment, two patients.

Treated within the Past Year and Still Alive—2/33

Two of the children treated during 1960 are still alive (August 1961), but one, with a primary in the left ilium, has developed secondaries. The other child, with a primary in the humerus, is still well. Treatment in each case consisted of local radiation.

D. Discussion

The depressing results reported are in general agreement with those recorded in the literature; indeed, in 1958, Harrison[10] reported that there were only 19 patients recorded in the English literature who have survived for more than 10 years after diagnosis and treatment of this tumour. Although the three patients in our series who have survived for over five years (26 years, 21 years and 7 years) have had no evidence of recurrence, late recurrences and delayed deaths from this tumour have been recorded.[6]

The argument as to the greater curative value of surgical as compared with radiation therapy cannot be resolved by this series. Of the three long-term survivors, two were treated surgically and one by radiation. In the large series reported by Dahlin, Coventry and Scanlon,[6] there is a suggestion that ablative surgical treatment is slightly better.

Surgical therapy for metastases has rarely seemed feasible in our group and was not employed. Several authors[6, 10] have reported isolated cases with prolonged survival after removal of pulmonary secondaries. The secondary tumour masses which appeared in the children of our series were treated by radiation, often with marked temporary relief of symptoms.

Recently, the chemotherapeutic agents have been of aid both at the time of biopsy to inhibit metastases and as palliative agents. Although one child with an apparently isolated tumour of the tibia did not respond to perfusion of the leg with massive doses of nitrogen mustard, several other drugs offer some hope for the future.

Summary

The clinical course of 33 children with Ewing's tumour of bone is reviewed and the pathological features are discussed. It would appear to be a distinct clinical and pathological entity among the malignant neoplasms of bone.

Two-thirds of the children were dead within one year of diagnosis. An assessment of the methods of therapy showed that either operation or radiation are occasionally curative. The five-year survival of three children (two for over 20 years) is noted. Failure in a single case where the isolated limb was perfused is also reported.

The authors wish to thank the members of the Surgical Staff at The Hospital for Sick Children for permission to review the charts of their patients. Our thanks are also due to Drs. W. L. Donohue, W. T. Mustard and R. B. Salter for reviewing the manuscript.

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RÉSUMÉ

Le sarcome d'Ewing est une tumeur qui apparaît dans les os des sujets jeunes, qui est très sensible aux traitements radiothérapiques mais qui est souvent mortel. Son origine est toujours discutée: à savoir si les cellules atteintes proviennent du tissu conjonctif médullaire ou de l'endothélium de sinusoïdes. Cet article étudie un ensemble de 33 cas de cette maladie traités à "The Hospital for Sick Children" de Toronto. Ces cas comprennent 20 filles et 13 garçons. Cette prépondérance des filles ne correspond pas à ce qui a été établi par des statistiques plus grandes. L'âge variait entre 13 mois et 16 ans. La symptomatologie ne présente généralement rien de caractéristique: enflure locale, mauvais état général, douleur locale vague, parfois un léger état fébril. Les examens de laboratoire ne montraient rien d'anormal. L'aspect radiologique classique est une lésion d'allure kystique avec réaction périostée. La biopsie est indispensable pour arriver à un diagnostic certain. Microscopiquement on trouve des cellules à contours peu nets ayant un noyau arrondi ou ovale, unique: Lorsque les phénomènes de nécrose ou de dégénérescence s'installent, les cellules peuvent prendre des formes polyhédriques comme l'avait décrit Ewing. Le traitement consiste en excisions locales ou radicales, en radiothérapie ou en chimiothérapie. Les resultats sont plutôt décevants. Sur les 33 cas présentés, trois eurent une survie de plus de cinq ans, deux de plus de trois ans et cinq de plus d'un an. Les méthodes modernes de chimiothérapie anticancéreuse qui ont été essayées n'ont été que peu prometteuses jusqu'à présent.

HARE-LIPS AND THEIR TREATMENT. A. B. LeMesurier. 169 pp. Illust. The Williams & Wilkins Company, Baltimore 2, Md., 1962. $7.00.

Whether one believes that the Hagedorn quadrilateral flap is the best method of cleft-lip repair is beside the point. It is a good method, sound in principle and has stood the test of time. It must be studied and re-studied by all those interested in this complicated deformity problem. Dr. LeMesurier's book is such a study. He appreciated the merits of the Hagedorn procedure in 1939 and adopted it in his practice at the Toronto Hospital for Sick Children. Here it has been used almost exclusively in the treatment of 1444 patients. This book is a detailed review of this experience and explains clearly the application of this modified operative procedure to all types of cleft-lip.

There are no fringe discussions. It is not a textbook and if one wishes chapters on history, genetics and anesthesia, one must look elsewhere. It is a meaty, concise, well-written and well-orientated volume of 169 pages. It pays little or no attention to the views or experience of others and has a small, yet pertinent bibliography. It deals strictly, yet generously, with the subject and method in hand. The photographs and diagrams are sufficient and excellent. The book is attractively printed and bound, and is offered at a reasonable price.

It is an excellent volume which can be recommended to all of those with interest or responsibility in this field.

THORACIC SURGICAL MANAGEMENT. 3rd ed. J. R. Belcher and M. F. Sturridge, with foreword by Sir Clement Price Thomas. 211 pp. Illust. Baillière, Tindall & Cox, London; The Macmillan Company of Canada Limited, Toronto, 1962. $5.40.

This relatively small book is directed toward those whose immediate responsibility is the preoperative and postoperative care of the thoracic surgical patient. The general principles of investigation and diagnosis are covered well in each section, and all the usual surgical problems are discussed. There is much less on tuberculosis, and much more on cardiac lesions in this edition than in previous ones.

This is a very practical handbook for the neophyte in thoracic surgery, and can be highly recommended.

ACUTE VOLVULUS OF THE CECUM

D. T. W. LIN, M.D., F.A.C.S.* and K. C. GRANT, M.D.,† *Montreal*

ACUTE cecal volvulus is an uncommon disease in which early diagnosis and treatment are often urgently required because it is associated with a high mortality. The first case of volvulus of the cecum was described in 1841 by Rokitansky. In order to distinguish volvulus of the cecum from volvulus of other parts of the colon and from complete volvulus of the mid-gut, Gatellier *et al.* in 1931 defined cecal volvulus as that condition in which torsion is confined to the cecum, ascending colon and terminal ileum.

INCIDENCE

A great variation in occurrence has been reported by different authors. Sweet[1] has cited the incidence as about 1.2% of all cases of bowel obstruction. About 11.3% of all cases of volvulus of the colon are found in the cecal area. The sex incidence is 3:1 in favour of males. It has been noted to occur most commonly in young adults, but rarely in infants. The mortality described in most of the older series has been in the range of 35%. This mortality increases sharply to over 70% in the presence of gangrene.[2]

CAUSAL FACTORS

The basis of cecal volvulus is failure of the normal fixation of the cecum and ascending colon in fetal life. This fixation normally takes place in the third stage of rotation following descent of the cecum to the right lower quadrant. The process by which the cecum loses its mobility and becomes attached to the posterior abdominal wall begins at about the eighth month of gestation and is complete four months after birth.

In 1942, Wolfler, Beaton and Anson[3] described 125 cadaver examinations of the ileocecal region and in 11.2% of these subjects an inadequate degree of fixation was present (Fig. 1) predisposing to volvulus.

Because the incidence of volvulus is in fact much lower than this figure (11.2%) would suggest, additional factors have been put forward as exciting causes. Among these are colonic distension; previous abdominal surgery; associated acute abdominal conditions, such as acute cholecystitis and perforation of the gallbladder; mesenteric adenitis; dietary extremes, i.e., a disproportionately high intake of gas-producing foods; intra-abdominal tumours, and pregnancy.

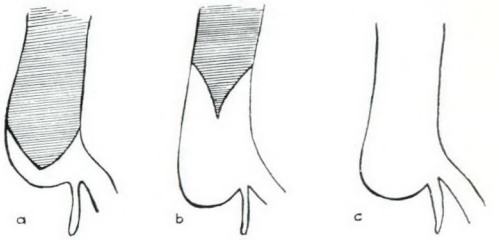

Fig. 1.—(a) Most of the specimens examined by Wolfler had sufficient peritoneal attachment to prevent volvulus; he described a wide range of normal attachments. Of Wolfler's specimens, 11.2% appeared predisposed to volvulus because of inadequate fixation. (b) The cecum and part of ascending colon is free of attachment to the posterior abdominal wall. [In (a) and (b) the shaded portions represent areas invested by peritoneum.] (c) A right colon entirely free of peritoneal attachment (from Wolfler *et al.*: *Surg. Gynec. Obstet.*, 74: 882, 1942).

CASE REPORT

W.O.K., a 53-year-old musician, began to feel crampy abdominal pain three days before his admission to the Royal Victoria Hospital, Montreal. He vomited several times and noticed an increasing distension of his abdomen. For two days before admission he passed no stool or gas.

His past history included an appendectomy in 1945 and a left upper lobectomy for pulmonary tuberculosis in 1961. At the time of his thoracotomy a congenital absence of the pericardium was discovered.

On physical examination, the patient was in no distress and moderately dehydrated. His blood pressure was 130/70 mm. Hg, pulse 95 per min., oral temperature 99.2° F. Abdominal distension was present. The bowel sounds were hyperactive. No muscle guard-

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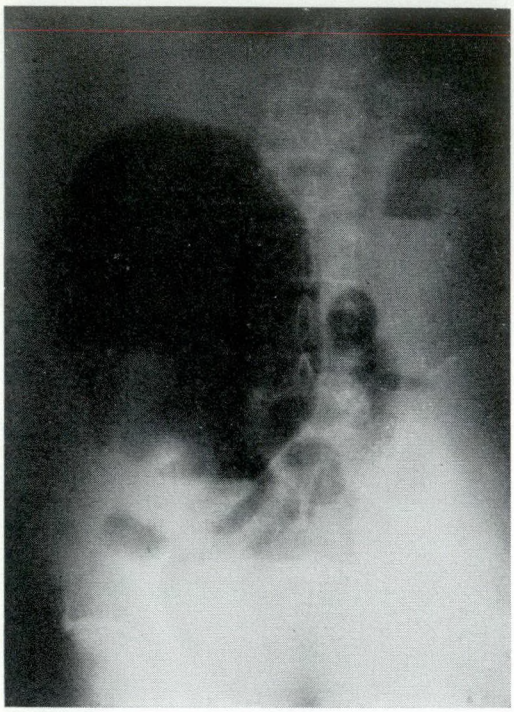

Fig. 2.—A hugely distended loop of bowel is noted in the right lower quadrant.

ing was present. Deep tenderness was elicited in the right lower quadrant.

The pertinent laboratory results were as follows: hemoglobin, 15.5 g. %; leukocyte count, 13,850/c.mm.; serum sodium, 129 m.Eq./l.; chloride, 87 mEq./l.; potassium, 4.67 mEq./l.

A flat plate of the abdomen revealed a hugely distended loop of bowel occupying the right lower quadrant; loops of distended small intestine seen proximal to this had fluid levels. No gas was present in the distal colon. A radiological diagnosis of volvulus of the cecum was made (Fig. 2).

After a brief interval necessary to allow rehydration and correction of electrolyte deficiencies, laparotomy was performed. An anticlockwise 180° volvulus of the cecum was found (Fig. 3). The volvulus included the terminal ileum, cecum and about 10 cm. of the ascending colon. A pencil-sized band extended across the ascending colon at the point of torsion and appeared to cause an exaggerated fixation of the colon at the junction between its fixed and mobile portions. The bowel was discoloured; some ecchymosis, but no infarction was present. The band causing the excessive degree of fixation was resected; a tube cecostomy and anterior cecopexy were performed.

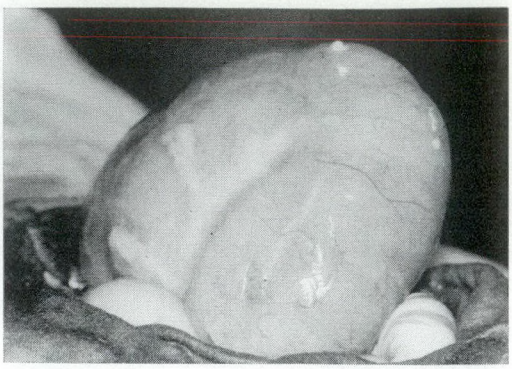

Fig. 3.—Volvulus of the terminal ileum, cecum and ascending colon at laparotomy. Note protrusion of dilated cecum and lower portion of ascending colon through wound. The dilated ileum is at the lower left.

The patient's postoperative course was complicated by pneumonia which responded to appropriate antibiotic therapy. The cecostomy tube was removed on the fourteenth postoperative day, and the subsequent fecal fistula closed spontaneously in four days. He was discharged from the surgical service 24 days after admission to the hospital.

Clinical Features

Three types of cecal volvulus were described by Gardiner[4] in 1947.

1. *Acute type.*—Fulminating and frequently associated with gangrene of the bowel.

2. *Subacute type.*—Simulating low small-bowel obstruction with a course of several days of abdominal pain, vomiting and gradual distension.

3. *Chronic type.*—Associated with recurrent episodes of right lower quadrant pain with spontaneous recovery. Barss *et al.*[5] in 1959 described 42 patients of this type whom they treated by cecopexy. The selection of these patients would appear to be difficult, but many patients with acute and subacute volvulus give a history of right lower quadrant pain.

Radiological Findings

In the literature, the correct preoperative diagnosis has appeared to depend upon accurate interpretation of the radiographs. A correct preoperative diagnosis was made in about 30% of reported cases.

Byrne, Swift and Farrell[6] in 1952 reported the typical radiological features of

TABLE I

	Case number	1	2	3	4	5	6	7	8	9	10	Average or %
Clinical features	Age	26	73	49	53	16	43	42	68	56	53	47.9 years
	Sex	F	F	F	F	M	F	M	F	M	M	60% female
	Duration of symptoms	5	2	½	2	1	1	3	6	1	2	2.4 days
	Presence of preoperative shock	—	+	—	+	—	—	—	—	—	—	20%
Operative findings	Clockwise rotation *v.* anticlockwise rotation	+	+	—	+	—	+	+	+	+	—	70%
	Presence of bands	+	—	+	—	+	—	+	—	—	+	30%
	Accurate preoperative diagnosis	—	—	—	+	—	—	—	—	+	+	30%
Operative treatment	Reduction only and lysis of bands	+		+		+		+				
	Cecopexy								+		+	
	Cecostomy						+				+	
	Resection		+		+					+		
Mortality					+							10%

cecal volvulus. A markedly distended loop of cecum and ascending colon was invariably present, usually assuming a transverse position in the right middle or upper abdomen and extending across the left. Associated with this was the loss of the normal cecal gas shadow in the right iliac fossa.

Occasionally a barium enema has been necessary for diagnosis, although this procedure is considered dangerous as a routine. Easton and Adams[7] have described obstruction of the barium column distal to the cecum with distended bowel just beyond as diagnostic of cecal volvulus.

Analysis of Cases

In the Royal Victoria Hospital, in the 30 years since 1932, 11 cases have been recorded. One of these was found at postmortem examination in a patient who had died from other disease; the volvulus was considered due to agonal causes. Only the acute and subacute cases were reviewed in this study because it was considered that the more numerous cases of chronic volvulus and "mobile cecum" did not lend themselves well to critical review.

Some of the pertinent features are set out in Table I.

Treatment

1. Bowel resection is universally agreed to be the only therapy for patients with gangrene; either a Mikulicz procedure or a one-stage resection should be carried out.

2. Reduction of the volvulus with anterior cecopexy or cecostomy is recommended by various authors. The latter accomplishes decompression as well as fixation of the bruised and damaged bowel.

Aspiration of the involved bowel before detorsion is recommended for patients in whom viability of the intestine is questionable. Rapid preoperative correction of fluid and electrolyte imbalance, and early operation is mandatory for all cases.[8]

Discussion

The relatively low mortality (10%) in this series is partly related to the relatively few cases of acute fulminating volvulus going on to gangrene. However, other writers[9, 10] have also described a lower mortality in the management of cecal volvulus. Improvements in surgical technique and the use of antibiotic agents have certainly contributed to this decline in mortality. In this review, no satisfactory clinical means was demonstrated of differentiating the acute and more lethal form of cecal volvulus from the subacute form. However, shock was present preoperatively in two of the cases; these proved to be associated with gangrene. None of the nongangrenous cases exhibited shock despite marked dehydration in several patients. Hinshaw, Carter and Joergenson[11] in 1959 noted that in abdominal "scout" films of

their acute cases (acute fulminating) a small bowel pattern did not have time to develop, while in the subacute cases (acute obstructive) the small bowel obstructive pattern became evident. We did not find this to be consistently true in our series.

In the cases reviewed, a notable incidence (five cases) of fibrous bands was noted in the operative reports. These bands appeared to result in excessive fixation of the ascending colon at the point of volvulus. They were the result of previous operation in only three of the five cases. In four of these five cases, the operative treatment was reduction of the volvulus only, with lysis of the bands causing fixation. No recurrence of volvulus occurred in any of these cases.

Summary

A review of cecal volvulus is presented with a description of 10 cases treated at the Royal Victoria Hospital, Montreal, over the past 30 years.

Attention is directed to a high incidence of fibrous bands causing excessive fixation of the ascending colon at the point of volvulus. It is suggested that this finding be sought specifically and recorded in such cases.

Although mortality in this condition has improved, the lethal character of cecal volvulus is underlined. The price of delayed operation may be the high mortality associated with intestinal gangrene.

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Résumé

Histoire d'un cas. Un homme âgé de 53 ans est admis d'urgence au "Royal Victoria Hospital" de Montréal pour un syndrome de douleurs abdominales remontant à trois jours auparavant. Depuis deux jours, le malade était en occlusion, n'ayant eu ni selle ni gaz. Il avait subi une appendicectomie en 1945 et une lobectomie supérieure gauche en 1961. L'examen montre un homme en état général assez bon, avec une température à 99.2° F., un pouls à 95. L'abdomen est ballonné, mais ne présente aucune contracture musculaire. Un cliché radiologique de l'abdomen à vide permet de voir une énorme anse intestinale distendue dans la région inférieure droite. On pose le diagnostic de volvulus du cæcum et l'intervention est effectuée peu après. On trouve un cæcum tordu dans le sens contraire des aiguilles d'une montre sur 180°, comprenant une partie de l'iléon terminal. Il existe une cicatrice large comme un crayon, rubannée causant une fixation exagérée du côlon au niveau de la jonction entre la partie libre et la partie fixée. Comme il n'y a pas de signes de gangrène, on pratique une cæcostomie avec fixation à la paroi. Dans les suites post-opératoires, on note une atteinte pneumonique qui réagit bien aux antibiotiques; le tube de drainage de la cæcostomie put être enlevé au quatorzième jour et la petite fistule stercorale subsistante se ferma spontanément en quatre jours. Le malade put être renvoyé chez lui 10 jours plus tard, guéri. Dans les annales du "Royal Victoria Hospital" on a pu retrouver une dizaine de cas semblables durant une période de 30 ans. Les auteurs insistent sur le haut degré de mortalité qu'entraîne cette affection; tout retard dans le diagnostic ou l'intervention, mène à la gangrène de la portion intestinale intéressée; les larges résections que nécessite alors la situation sont toujours graves.

FACTORS IN THE IMPROVEMENT OF RESULTS IN VARICOSE VEIN SURGERY*

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THE standard and approved method of treatment of incompetent varicose veins is high ligation and stripping of the great and/or lesser saphenous systems and their major branches. We have employed this method since 1950 and, with the experience and follow-up study of well over 1000 cases, have gradually modified our technique so that our results have consistently improved.

This is a difficult abnormality to cure permanently by operation. The hereditary defect in the superficial veins of the legs remains and continues to affect progressively any major veins or branches left behind at operation; hence, so-called recurrences take place. For this reason, there is great need for proper assessment of each case to ensure that all incompetent veins in the involved leg are removed; this condition also requires expert and complete surgery to ensure that all major branches of the vein are dealt with.

The purpose of this article is to describe certain of our modifications of vein stripping in order to present some ideas which may help other surgeons working in this field to better their results.

The reason for poor results in varicose vein surgery are many. Some of these are listed below.

Reasons For Poor Results in Vein Surgery

1. Improper assessment with respect to what veins should be stripped, thereby missing an incompetent lesser saphenous or perforating vein.
2. Errors in the proper identification of the saphenofemoral junction and therefore performing a ligation too far distally.
3. Distal ligation at a point ½″ to 1″ below the junction and neglecting the upper saphenous tributaries.
4. Failure to strip all major branches.
5. Failure to look for a saphenous reduplication.
6. Failure to expose locally every mass of veins which may indicate a "perforator".
7. Failure to explore, strip or excise every vein of any consequence exposed by these local explorations.
8. Failure to pass the stripper both from above down and below up in every case.

ASSESSMENT OF CASES

A careful examination and history-taking will rule out associated conditions such as osteoarthritis of the knees, flat feet and various neurological lesions which are the true cause of symptoms for which a few harmless veins are blamed.

We employ the Trendelenburg test and its modifications to determine which veins are incompetent. This test will demonstrate an incompetent valvular mechanism and therefore the indication for surgical therapy. However, the presence of incompetent lesser saphenous or perforating veins may not be obvious. The lesser saphenous, if incompetent, can almost invariably be palpated as a softish cord in the popliteal space and frequently down the calf. Males seem more prone to lesser saphenous incompetence than females. A cluster of dilated veins about the lateral malleolus should also raise suspicion of the presence of this condition. Occasionally, this cluster is due to varices in the lower lesser saphenous, originating from incompetence of the large connector branch in the medial calf between the greater and lesser saphenous systems. Perforating veins between the superficial and deep systems, when incompetent, usually show themselves by an abnormal cluster of superficial veins in the area. Therefore, any such cluster warrants a separate incision not only for their excision but for search for the "perforator". The total assessment of each case must be reviewed by the surgeon in written notes,

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which should be read again just before the operation. We do not believe in preoperative skin-marking of veins. If there is any doubt as to vein branches when the patient is on the operating table, placing him in the reverse Trendelenburg position for a few minutes will indicate these involved tributaries.

Operative Details

Identification of the saphenofemoral junction should not be difficult, but frequently the saphenous vein branches immediately in a reverse Y and one limb of this Y may be mistaken for the saphenous and the other limb of the Y for the common femoral, an error which results in a major portion of the saphenous being left behind. The ligation should be flush with the common femoral vein above all tributaries. We now make it a practice to retract the lower edge of the groin incision downward and dissect the saphenous distally with the fingers before stripping. In this way, large major branches which require stripping can be identified. The frequently occurring lateral descending branch that travels laterally across the anterior thigh passing above the patella and down the lateral calf can be identified in this manner. The same applies to the medial descending branch which winds from medial to lateral across the posterior thigh.

However, more important than the above-mentioned possibility of error with respect to the saphenous branches is the fact that in nearly 50% of cases an accessory saphenous vein (reduplication) will be found arising anywhere in an area as far as 6″ distal to the saphenofemoral junction. This accessory saphenous parallels the main saphenous vein within an inch and is usually more superficially placed. The finding of superficial medial thigh varices is a certain indication of the presence of such a branch. The main saphenous trunk is relatively deeply placed in the fat of the thigh and rarely gives rise to varices which are seen superficially. This accessory saphenous vein may peter out in small branches in the calf or may continue down to the ankle. Obviously, leaving these major radicals behind will give a poor surgical result.

Stripping should be performed both from above and below. When passing it down from above, the stripper should be guided into one of the major calf branches as far as it will go.

The below-ankle incision is then made one finger's breadth below and anterior to the medial malleolus. At this location three moderate-sized terminal tributaries will be found issuing from the saphenous, two heading toward the heel and one to the anterior aspect of the ankle. Dissection and terminal avulsion of as much as possible of these branches should be done to eliminate the myriads of little spider skin venules that occur so frequently in this area. The stripper is then passed upward and may traverse an entirely separate main vein than the downgoing stripper. This separate vein is usually a reduplicated saphenous vein referred to above.

Any local cluster of varices should be explored by a separate incision. Excision of these clusters is carried out and frequently unsuspected major branches leading anterior or posterior are found which can be stripped. In this way also, perforating veins are found penetrating directly inward through the deep fascia.

There are many varices in branch veins which cannot be stripped because of the tortuosity of the vein and the thinness of the varices; the distal ends of the medial and lateral descending branches are of this nature. However, these should be dealt with at time of operation and not left for postoperative injection therapy. Our method in this type of varix is to fragment these branches through a series of small step-ladder incisions along the course of the branch about 12″ apart. A thin long-handled pair of scissors is inserted along the course of the branch and the varices are macerated by rapid cutting with the scissors. These macerated veins are obliterated by thrombus and later by scar tissue; any bleeding at operation is easily stopped by pressure. We employ this same technique to obliterate nests of little cutaneous spider veins that are otherwise resistant to treatment. With the curve of the scissors turned toward the skin from beneath, the connection of these spider veins to their subcutaneous feeders are severed. Complete

disappearance of all spiders may not take place but the majority will be obliterated if the procedure is adequately done.

The lesser saphenous vein has never been reduplicated in our experience and can usually be stripped *in toto*. If difficulty is encountered in finding the vein in the popliteal space, it can be found behind and below the lateral malleolus; the stripper when passed from below points the way to its location in the popliteal space.

Discussion

As previously mentioned, the long-term results in varicose vein surgery will depend on the carefulness and extensiveness of the operation performed. A few branches usually remain in the postoperative period and these are dealt with by local sclerosing injections. With passing years, our experience is that the majority of cases will require one to two postoperative injections. Other small branches will dilate subsequently and begin to show and hence we advise patients who have undergone vein surgery to report every year for review so that any of these new branches can be injected.

Additional Indications for Vein Surgery

The uncomplicated hereditary type of varicose vein is by far the most common type. However, complications of varicose veins are frequent and these will be referred to briefly.

Secondary varicose veins occur years after in many cases of previous deep thrombophlebitis. The history, the appearance of the leg, and, if necessary, venography will confirm the damage caused by the deep phlebitis. These secondary varicose veins can be large, with incompetent valves, and constitute an added insult to injury which occurs owing to venous stasis of the leg when the patient is upright. Most authorities state that because of damage to the deep venous system, these superficial veins should not be disturbed. However, when the Trendelenburg test is positive in these patients, we feel that the usual vein stripping should be carried out to reduce the added venous stasis produced in the standing position by these superficial veins. The patient is warned that this procedure will just help and not cure his condition and that he must continue to look after the postphlebitic leg by leg elevation and use of an elastic stocking. Stripping is frequently of sufficient benefit to convert a postphlebitic leg from one which has been difficult to manage, because of complications such as eczema, indurations and ulceration, to a leg which is easily managed by the continuing conservative regimen. Two further points should be emphasized; namely, no superficial vein operation should be carried out unless these veins are grossly incompetent. One so often sees saphenous vein ligations and stripping performed upon patients when these veins were still competent. This is bad surgery and leads to no improvement. The second point is that in these cases of secondary varicose veins, the number of incompetent communicating veins is abnormally large; these should be most carefully searched for and ligated.

Our therapy of acute superficial phlebitis in varicose veins has undergone considerable modification. When a few hard tender lumps are seen below the knee, with redness and induration, we are not unduly concerned and treat the patient with elastic support and continued ambulation. Future arrangements should be made to have these veins stripped. However, when the thrombosed varices are extensive and especially where the thrombosis is spreading up to the saphenofemoral junction, then a more serious view should be taken of the situation. We have many authenticated cases where the superficial thrombus has extended into the common femoral vein at the saphenofemoral junction producing embolization or a deep phlebitis. When the phlebitis is extending toward the groin, such a patient should have an operation as a semi-emergency, with the usual high ligation as the first step. The thrombus has usually stopped at the edge of the fossa ovalis about half an inch from the junction, but on rare occasions will have extended into the femoral. In the latter eventuality, removal through the saphenous stump should be attempted, but often the thrombus is adherent to the vein wall and cannot be removed easily.

In such an instance, immediate full anticoagulant therapy is indicated postoperatively, relying on heparin entirely for the first five to seven days. After ligation, we believe a regular stripping procedure should be performed by stripping or excising the thrombosed varices. The rationale here is that the stripping removes the primary cause for the superficial phlebitis, namely the varicose veins and also the large tender thrombosed lumps, which otherwise take three to four months to disappear and are accompanied by attendant disability. Hospitalization in the average case is no longer than in the uncomplicated vein case.

In the other types of complication resulting from varicose veins, we treat any ulceration or eczema by conservative therapy, using Unna's boot compression before carrying out vein operation. We do not like to perform vein operation on the pregnant woman unless extraordinary circumstances are present. The patient is advised to wait for from six to 12 weeks postpartum before stripping is carried out.

Summary

The authors' current techniques in the surgical treatment of saphenous varicose veins have been reviewed. The indications for high ligation and stripping have been broadened to include operation in cases of superficial thrombophlebitis and, in addition, the application of surgery in secondary varicose veins ensuing after deep thrombophlebitis has been noted.

Methods for minimizing the recurrence of varicose veins have been emphasized and these include the careful removal, based on an adequate preoperative assessment, of all significant venous channels with reduction to a minimum of postoperative injection therapy. The many variations encountered in the superficial venous system have been emphasized and the operative technique has been revised so as to more adequately manage these variations. Meticulous surgery for varicose veins will provide rewarding cosmetic and symptomatic relief in the majority of patients. Major operative complications should not occur if the principles outlined above are followed.

Résumé

Les méthodes de traitement qui sont les plus utilisées dans le traitement de varices sont la ligature haute et le "stripping". Les auteurs emploient ces deux méthodes depuis plus de dix ans et leur expérience, qu'ils présentent ici, repose sur plus de 1000 cas. Ils constatent qu'après ces interventions, il existe un pourcentage de récidive: ceci est dû en premier lieu au fait que le patient porteur de varices est indiscutablement prédisposé à cette affection. Cependant il est certains points techniques qui peuvent permettre d'améliorer les résultats éloignés. Les principales fautes à éviter sont les suivantes: mauvaise ou incomplète orientation anatomique; erreurs dans l'identification de la jonction saphéno-fémorale entraînant une ligature trop basse; "stripping" incomplet laissant subsister des branches; négligence dans le passage du "stripper" de bas en haut et de haut en bas; dissection incomplète des paquets variqueux correspondant à une branche perforante. Les veines variqueuses superficielles peuvent être causées par une thrombophlébite profonde: la veinographie permet de faire ce diagnostic. Classiquement, il est admis que, dans de tels cas, l'opération des varices est contre-indiquée; les auteurs estiment cependant que, si le test de Trendelenburg est positif chez ces malades, cela suffit à créer une indication opératoire et qu'alors on doit procéder à un "stripping". En effet, on améliorera ainsi leur circulation de retour. Lorsqu'on se trouve en face d'une phlébite superficielle aiguë, le meilleur traitement sera conservatif: il n'en est pas de même si l'on s'aperçoit que le processus phlébitique s'étend et remonte vers la jonction saphéno-fémorale. A ce moment, on se trouve devant un état de semi-urgence et la ligature doit être pratiquée.

ANATOMICAL STUDIES OF THE AMPULLA OF VATER

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PANCREATITIS remains a disease of uncertain etiology despite much medical endeavour. Sedation, alimentary rest, depression of pancreatic secretion, restoration of blood volume and later surgical attack upon associated biliary disease are the mainstays of management. Following the recommendation that division of the sphincter of Oddi should be carried out to prevent recurrence of pancreatitis,[9, 13] the detailed anatomy of this passage formed by the junction of the common bile duct and the main pancreatic duct has assumed increased surgical significance. For the sake of simplicity in this paper, this passage shall be designated as the "common channel", draining both bile and pancreatic juice into the duodenum.

Descriptions of the ampulla of Vater in the standard anatomy texts[18] leave something to be desired by the surgeon about to undertake a sphincterotomy, as attested by the voluminous literature upon this subject. The percentage of cases in which a common channel is found has been reported from 9%[16] to 89%.[21, 23] With such variation in mind, a further investigation was undertaken. Several specimens have been photographed to illustrate the usual anatomy and some of the variations. An attempt has also been made to determine how many of these specimens might be obstructed by a stone in the common channel which would allow reflux of bile into the pancreas as originally described by Opie in 1901.

MATERIALS AND METHOD

One hundred and twenty-five specimens were obtained during routine postmortem examinations at Westminster D.V.A. Hospital, London, Ontario. The patient population is almost exclusively male and elderly. There were no examples of pancreatitis.

Following preliminary trials of corrosion cast technique, meticulous gross dissection was found most satisfactory. With non-fixed tissues, it is easily demonstrated that a short common channel can be made to appear as a papilla with separate openings for the pancreatic and bile ducts. All specimens were fixed in 10% formalin before dissection to avoid such artefacts. Optical aids were used in some cases.

Histological preparations did not contribute additional information.[20] Radiological demonstrations were less useful than gross dissection.[22]

OBSERVATIONS

TABLE I.—INCIDENCE OF COMMON CHANNEL

	No. of specimens	%
Common channel	90	72
Separate openings	35*	28
Total specimens	125	

*In four cases the openings were more than 1 cm. apart.

In 30 specimens both ducts opened on the papilla of Vater.

Careful dissection and inspection of 125 autopsy specimens of the pancreas, fixed in formalin without traction or distortion, indicate that three out of four have a channel common to the biliary and pancreatic duct systems (Table I).

In no case was this common channel enlarged or fusiform. Fixation would diminish the calibre of the original lumen.

TABLE II.—LENGTH OF COMMON CHANNEL

	No. of specimens	%
2 mm. or less	42	47
3 mm. - 5 mm.	32	35
6 mm. - 9 mm.	5	6
10 mm.	9	10
Not measured	2	2
	90	100
Mean	3.8 mm.	

Note that in 82% of the cases in which a common channel was found, this was so short and thin-walled that stone impaction would be most unlikely.

Only nine of 125 specimens (7%) had a common channel 1 cm. long in which impaction of a stone would be likely to produce bile reflux into the pancreatic duct.

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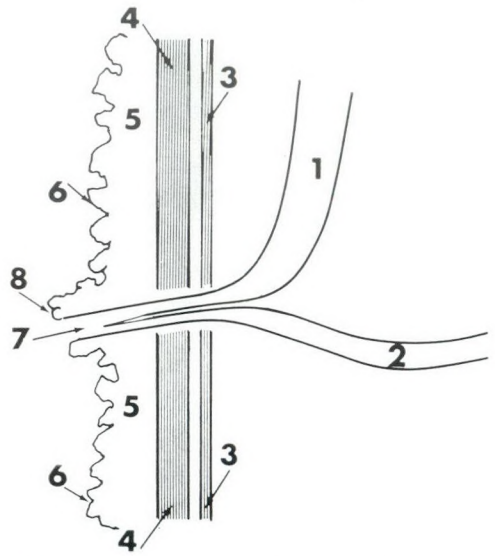

Fig. 1.—This diagram illustrates the common pattern. 1. Common bile duct. 2. Pancreatic duct. 3. Longitudinal muscle of duodenum. 4. Circular muscle of duodenum. 5. Submucosa. 6. Mucosa. 7. Common channel. 8. Papilla of Vater. Note the short common channel and the position of both ducts in the submucosa of the duodenum.

TABLE III.—Associated Pathology

Gallstones—with common channel	15 (16.7%)
with separate channels	6 (17.1%)
Total	21 (16.8%)
Cirrhosis	3
Liver cyst	2
Pancreatic cyst	2
Pancreatitis	1
Periampullary diverticulum	1

There is no significant difference between those cases with a common channel and those with separate openings.

Approximately four-fifths of these common channels were less than 5 mm. in length and only nine exceeded 1 cm. in length. Fourteen of these 125 specimens fulfilled Opie's criterion for reflux (5 mm. long) (Table II).

Figs. 2, 3, 4 and 5 are photographs of specimens illustrating the common anatomical pattern. Fig. 1 is a diagram of the same. Note the shortness of the common channel, the thinness of the partition between the common bile duct and the pancreatic duct, and the position of the common channel in the submucosa of the

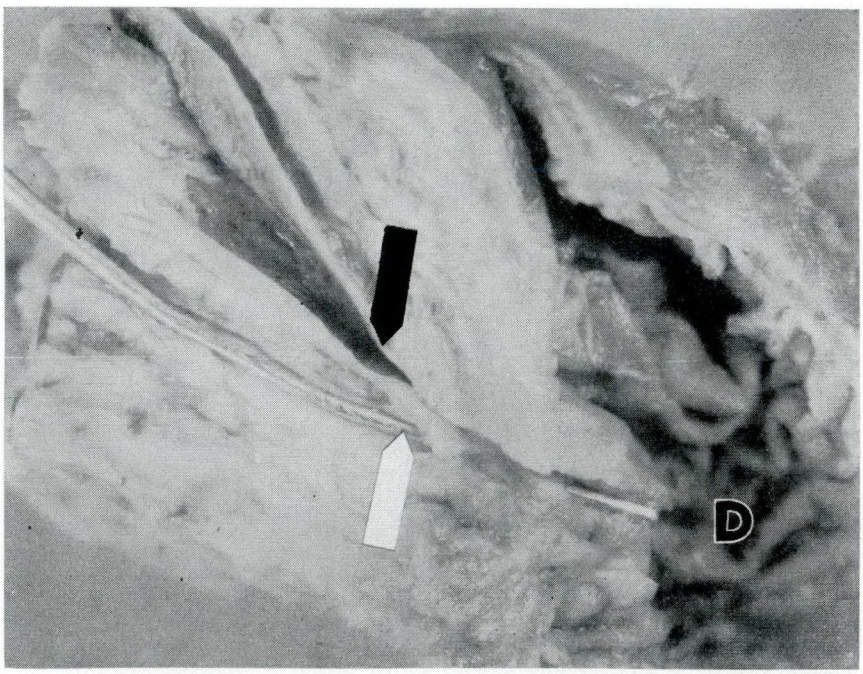

Fig. 2.—Common channel 4 mm. long. The probe lies in the pancreatic duct (Wirsung). In the illustrations, the black arrow indicates the common bile duct, the white arrow the main pancreatic duct and the letter D the duodenum. The dissections are viewed from the dorsal aspect.

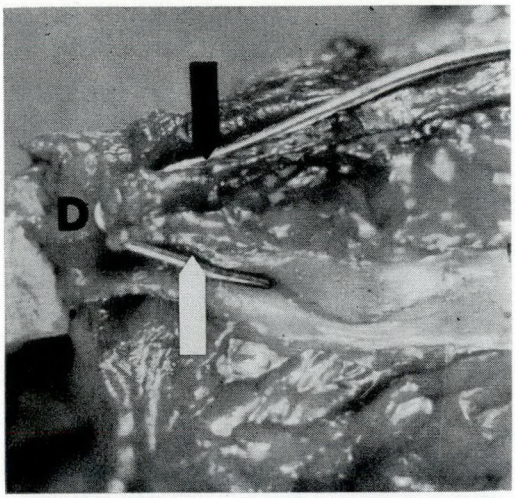

Fig. 3.—Common channel 4 mm. long. The stem of the probe lies in the common bile duct and the tip is bent to lie in the pancreatic duct.

duodenum. The footnotes to the tables and figures are self-explanatory.

Fig. 6 demonstrates a gallstone impacted in the common bile duct outside the duodenal wall. At this site, the lumen diminishes, the duct angulates and perhaps probes in the common duct are arrested here.

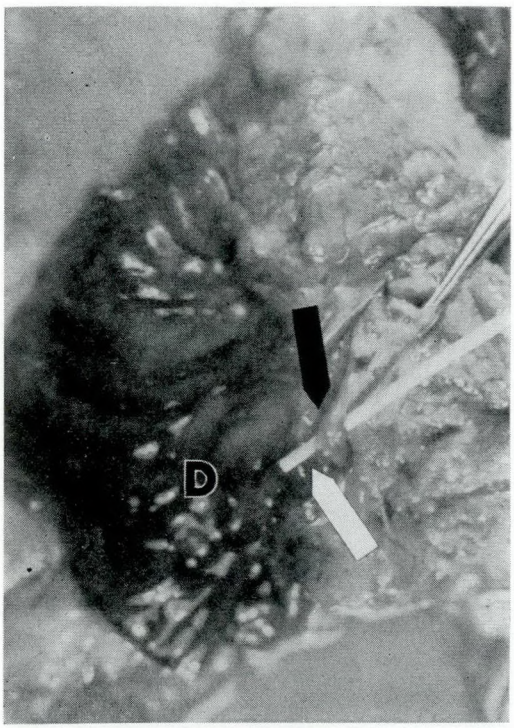

Fig. 4.—Common channel 6 mm. long. The probe lies in the pancreatic duct.

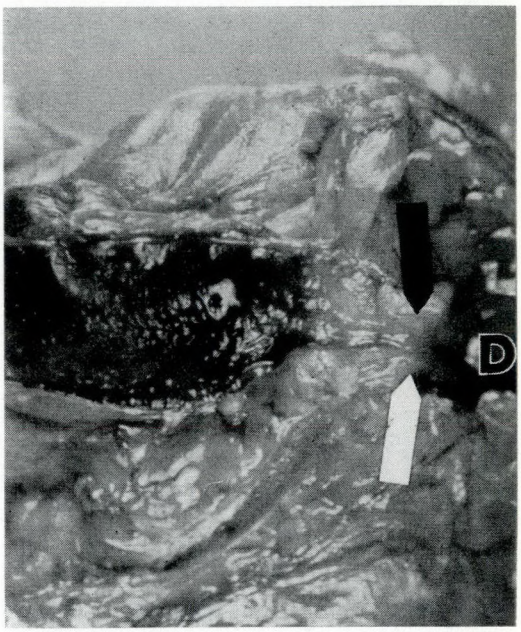

Fig. 5.—Common channel 4 mm. long. Note gross dilatation of the common bile duct and a small stone outside the duodenal wall.

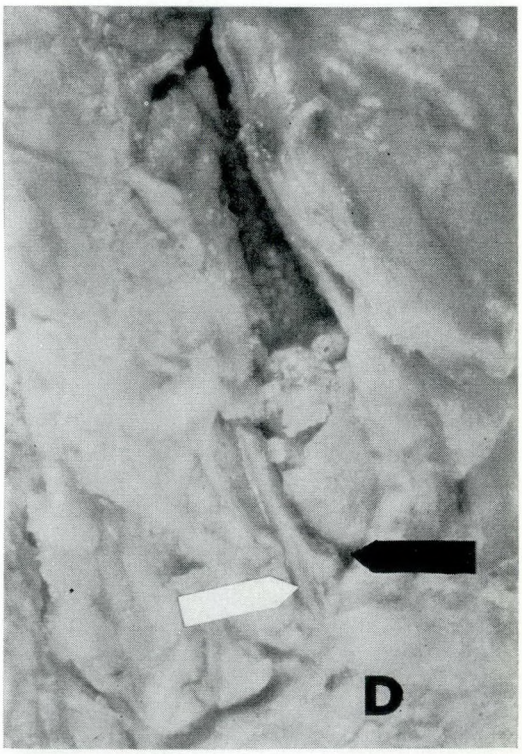

Fig. 6.—Common channel 1 mm. long. The gallstone measures 1.5 cm. x 1 cm. and lies outside the duodenum.

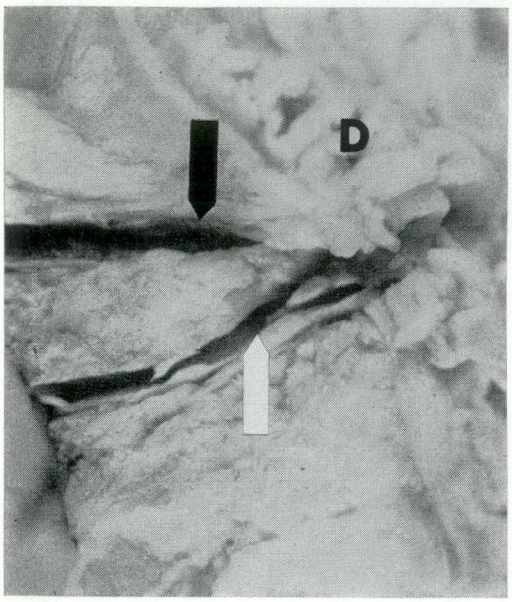

Fig. 7.—Separate duct openings on duodenal papilla.

Figs. 7, 8 and 9 exemplify ducts which open separately. A stone in the bile passage might distort the thin partition and obstruct the pancreatic duct.

Fig. 10 illustrates the less common type in which the duct openings are widely separated. In this specimen, the dominant pancreatic duct is the duct of Santorini.

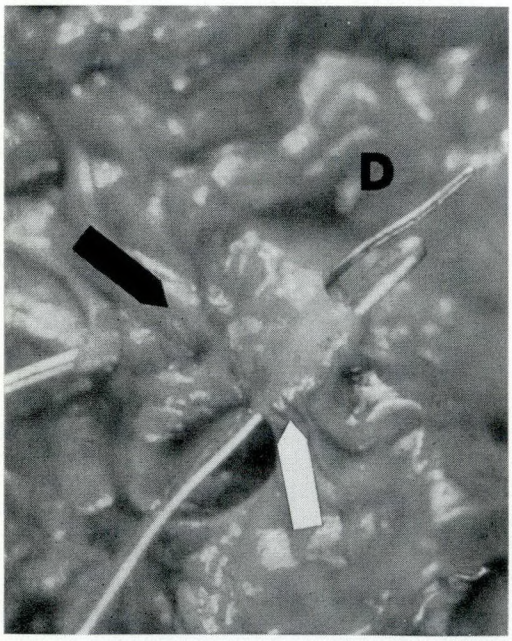

Fig. 8.—Separate openings on duodenal papilla.

Fig. 9.—Separate openings on duodenal papilla. Note the large accessory pancreatic duct (Santorini) and the thin partition.

Discussion

An ampulla is a flask-like dilatation.[8] This configuration was not observed in these 125 specimens. One questions the use

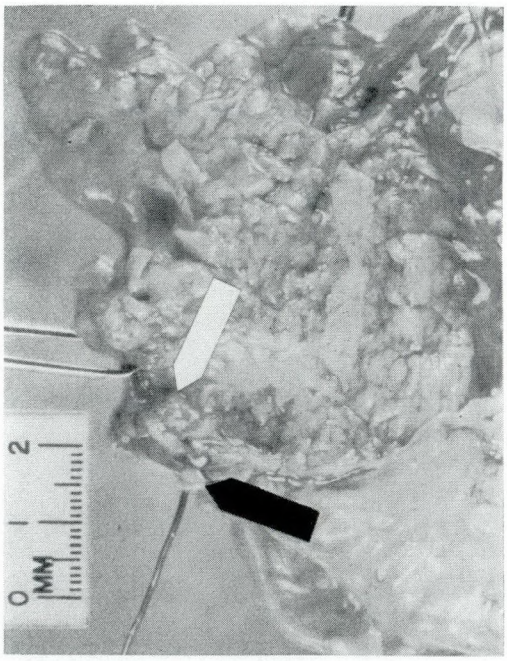

Fig. 10.—Ducts opening 2 cm. apart. The probe lies in the common bile duct.

of this term as applied to the common channel.

Duct obstruction,[23, 24] vascular obstruction,[1] enzyme activation,[12, 17] infection and auto-immunity[27] are all factors to be considered in the pathogenesis of pancreatitis. This study demonstrates that a common channel is found in 72% (Table I) of cases but in 82% of these the common channel was 5 mm. or less in length. Accepting Opie's opinion that 5 mm. was the shortest length in which a stone might impact and produce reflux of bile into the pancreatic ducts, only 14 (11%) of these specimens fulfilled this condition (Table II).

The partition separating the bile duct from the pancreatic duct is frequently a thin fold, much like a curtain. It is possible that a stone in the common bile duct might obstruct the pancreatic duct because they run parallel in the submucosa of the duodenum.

The influence of the duodenal wall upon the duct systems has probably been underrated. The common duct turns and diminishes in size as it penetrates the medial wall of the duodenum. The point of entry of the common duct into the duodenal wall is a place where sounds may frequently stop. The duct of Wirsung runs dorsal to the common bile duct, curves gradually and diminishes in size. We have observed stones lodged just outside the duodenal wall (Fig. 6). Duodenal diverticuli occur adjacent to the pancreatic duct and may obstruct it.

Wisniewski, Williams and MacKenzie[29] have emphasized the importance of duodenal obstruction in the production of pancreatitis.

Doubilet[9, 10] has stated that a common channel occurs in those patients who have pancreatitis. Since all specimens in our series were normal we have no data to support or to refute this thesis.

In 34% of cases the common channel was 2 mm. or less in length. Since this circumferential thin mucosal fold is easily pushed back, perhaps this accounts in part for the great discrepancy in the incidence of a common channel reported in the literature, particularly when fresh specimens are used. Traction on the ducts,[9] which may occur in radiological demonstrations, may also distort the ducts. The figures given above agree closely with those reported by Dowdy *et al.*,[11] Hermann and Davis,[14] Opie[23] and Baggenstoss.[2]

Sphincterotomy has become a popular operation.[28] In these specimens, a 1 cm. incision would create a separate opening into the duodenum in 90% of cases with a common channel (Table II).

Summary

One hundred and twenty-five formalin-fixed specimens of the duodenum and pancreas have been dissected in detail. The figures illustrate the anatomy.

A common channel for the pancreatic and biliary duct systems was observed in 72%. No dilatation of this channel was observed. In all cases the common channel lay in the submucosa of the duodenum and opened at the tip of the papilla of Vater. The mean length of the common channel was 3.8 mm. and it exceeded 5 mm. in only 14 cases.

Gallstones were recorded in 17% of these cases and were not related to the presence or absence of a common channel.

The suggestion is advanced that the common site for impaction of a stone in the common bile duct is at the point of entrance into the duodenum.

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Résumé

Les problèmes étiologiques que posent certaines affections comme la pancréatite, ont amené le monde médical à revoir de plus près l'anatomie du sphincter d'Oddi et de l'ampoule de Vater. L'auteur a entrepris des recherches personnelles sur cette question en utilisant 125 spécimens provenant d'autopsies de routine effectuées au "Westminster D.V.A. Hospital", London, Ontario. Il fut fait quelques essais de moulage plastiques par corrosion, mais la dissection soigneuse se révéla plus profitable. Il a pu être démontré que dans trois cas sur quatre, il existe un canal commun qui sert de confluent aux canaux cholédoque et pancréatique. Ce canal est cylindrique, sans dilatation, contrairement à ce que l'on pourrait croire: le terme "ampoule" désigne en effet un objet renflé ressemblant plus ou moins à une bouteille; une telle configuration n'a pas été trouvée dans les spécimens étudiés. Ce canal commun est très court, et les canaux cholédoque et pancréatique y débouchent très près l'un de l'autre après avoir effectué un trajet presque parallèle pendant une certaine distance: ceci peut expliquer qu'un calcul du cholédoque soit capable de provoquer un écrasement du pancréatique et un trouble important dans l'écoulement du sac pancréatique. Le canal commun traverse la sous-muqueuse duodénale et vient s'ouvrir à l'extrémité de la papille de Vater. L'auteur conclut que le point d'enclavement le plus fréquemment rencontré pour un calcul biliaire est l'orifice duodénal.

MULTIPLE FAMILIAL POLYPOSIS OF COLON WITH ASSOCIATED MANIFESTATIONS*

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MULTIPLE familial polyposis of the colon was first described by Harrison Cripps in 1882. Lockhart-Mummery and Dukes[6] classified and stressed the hereditary aspects of this disease.

From these and other studies the following facts have been established and generally recognized. The disease starts as a genetic mutation. It is transmitted to succeeding generations as a Mendelian dominant so that only one-half of the children are likely to inherit the disease. It is non sex-linked, involving males and females equally, and both sexes may transmit the abnormality. Only those with polyposis may transmit it. Virtually all of those affected will develop carcinoma and at a younger age than the average for carcinoma of large bowel. The carcinoma is not inherited but rather a propensity of the colon mucosa to undergo epithelial hyperplasia. The polyps are not present at birth, but usually make their appearance about puberty and the average age of onset of symptoms is 20 to 30 years.

ASSOCIATED MANIFESTATIONS

While the basic characteristic of the disease is the presence of innumerable sessile or polypoid adenomata involving the mucosa of the entire large bowel, a less generally recognized feature is the frequent simultaneous occurrence of bony and soft tissue tumours.

TABLE I.—GARDNER'S SYNDROME

Multiple diffuse polyposis	
Osteomatosis	
Soft tissue tumours	Sebaceous cysts
	Fibrous tissue tumours

In 1951, Gardner and Richards[1] reported a family with a syndrome consisting of multiple polyposis, multiple osteomata and multiple soft tissue tumours (Table I). The latter are chiefly sebaceous cysts and fibrous tissue tumours. These fibromas may be present subcutaneously or, as subsequent reports[2-4] have shown may be found in the retroperitoneal spaces, as masses in the mesentery and particularly in healed surgical incisions where they appear as typical desmoids.[5] The osteomata are found chiefly in the skull and facial bones.

The unusually high incidence of these additional features in polyposis patients is best explained by a tendency to excessive proliferation of cells of mesenchymal origin, and Gardner and Richards[1] have postulated that a single defective gene arising as a mutation may be responsible.

MATERIAL

This report deals with two unrelated families in which the typical features of polyposis were present and in whom a number of associated mesenchymal tumours were demonstrated. In *Family 1* every member of the family of six siblings was affected, an unusually high degree of involvement. The mother died at the age of 35 from carcinoma of the liver which may well have been metastatic from a large bowel carcinoma. The family history so far as can be traced is otherwise negative. Four of the siblings were female and two male; their ages at time of diagnosis ranged from 22 to 31 years.

Only one, a 26-year-old woman, had symptoms sufficient to require medical advice, these being crampy abdominal pain and bloody diarrhea of one year's duration.

Diagnosis was established in all cases by sigmoidoscopy and barium enema. None had invasive carcinoma.

This family displayed a noteworthy number of associated soft tissue and bony lesions of the type described by Gardner. A representative section of large bowel showing the multiple adenomata may be seen in Fig. 1. Typical soft tissue tumours are seen on the back (Fig. 2) and face (Fig. 3) of the same man. The asymmetry

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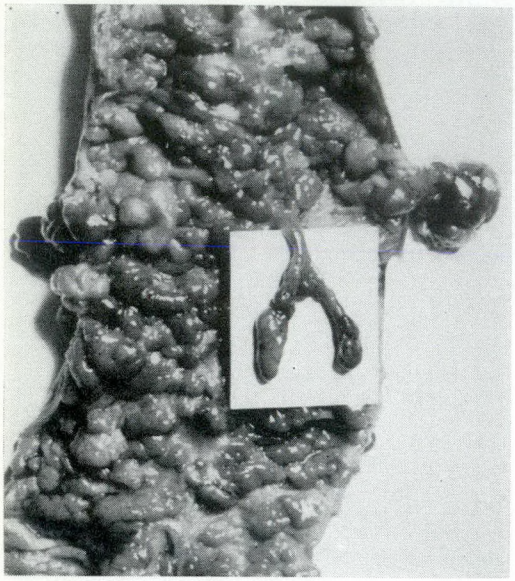

Fig. 1.—Representative segment of large bowel showing multiple polypoid adenomata.

noted over the right mandible in Fig. 3 is an osteoma clearly visible in the radiograph (Fig. 4) and a film of his skull (Fig. 5) shows numerous broad-based flat osteomata involving the outer table. Examination, including skeletal survey, of the other

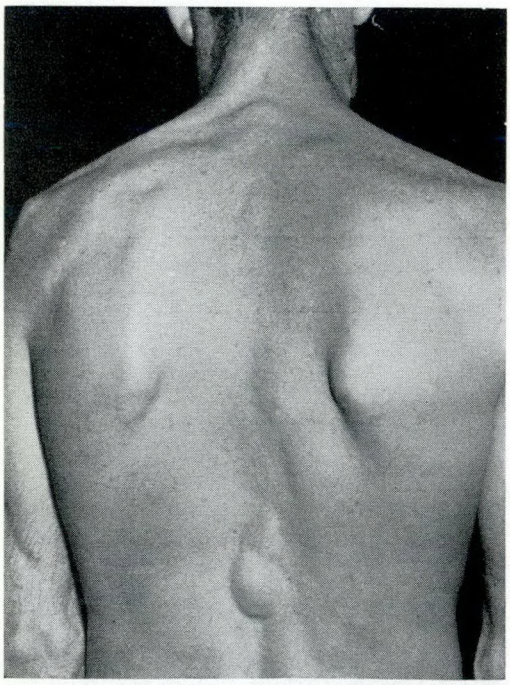

Fig. 2.—Soft tissue tumour in middle of back. Same case as Fig. 1.

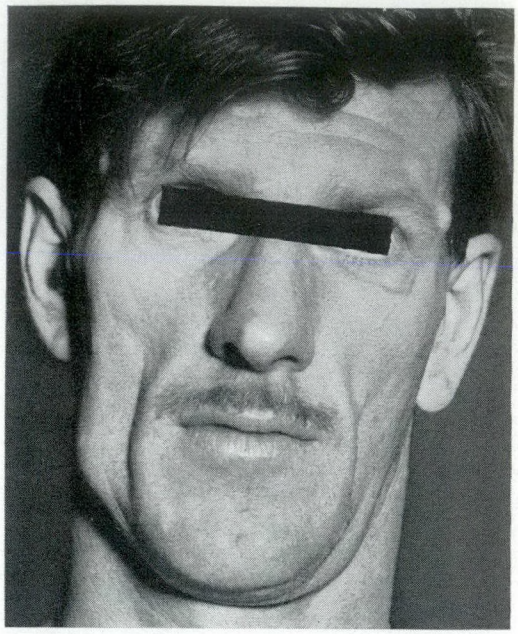

Fig. 3.—Multiple soft tissue nodules (sebaceous cysts) about left eye. Note asymmetry of right mandible. Same case as Fig. 1.

members revealed that the entire group displayed one or both of these associated lesions (Table II). Three showed the complete triad of Gardner's syndrome. Two

TABLE II.—Polyps and Associated Lesions in Family 1 Showing Relationship of Polyps to Associated Bony and Soft Tissue Tumours

	Polyps	*Osteomata*	*Soft tissue tumours*
K.P.	yes	yes	yes
M.H.	yes	yes	yes
R.W.	yes	yes	no
D.P.	yes	yes	no
E.S.	yes	yes	yes
B.W.	yes	no	yes

showed polyps plus osteomata and one showed polyps plus soft tissue tumours. In all instances the soft tissue tumours were epidermoid cysts and the osteomata were all limited to the skull and/or facial bones.

The six siblings have collectively produced 13 children to date, the eldest of whom is 10 years of age, so that investigation except in isolated instances has not yet been undertaken. Eight of these children are those of one member of this family and it is of some interest to note that three of the eight died at the age of three months of generalized glycogen storage disease.[11]

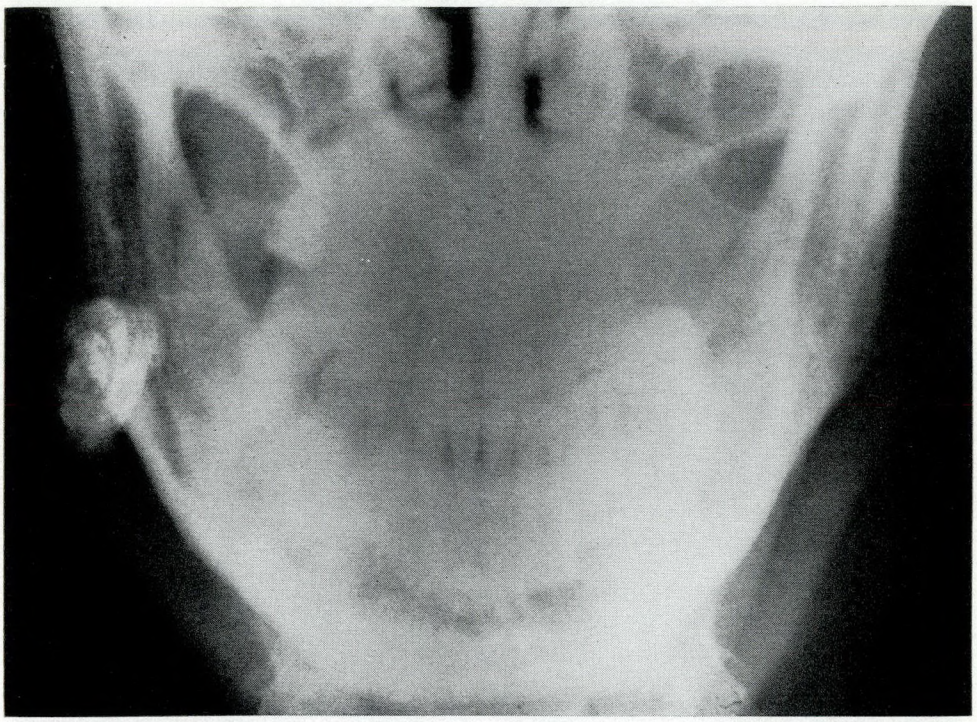

Fig. 4.—Radiograph showing osteoma of right mandible.

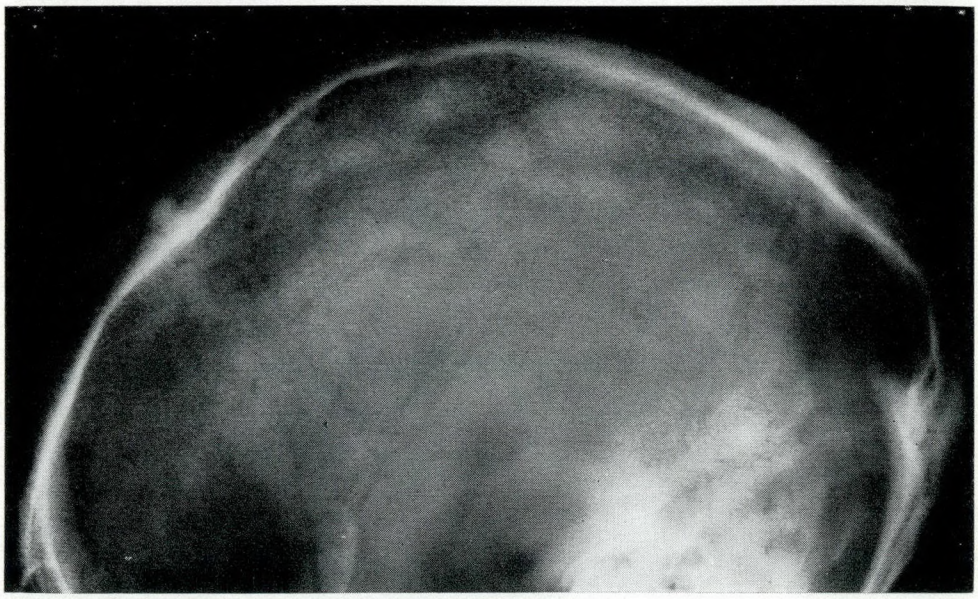

Fig. 5.—Radiograph of skull showing osteomata of outer table.

Family 2.—Five out of eight were affected; all were males and ranged in age from 23 to 36 years at the time of the diagnosis. Familial involvement could be traced back two generations on the maternal side. In contrast to *Family 1,* all had symptoms of bloody diarrhea for periods of up to two years before seeking advice. Two had invasive carcinoma when first seen and died of the disease. The remaining three showed no evidence of invasive malignancy and are well to date.

Fig. 6.—Desmoid tumour from rectus muscle of patient, R.T., showing infiltration of fibrous tissue between muscle fibres.

On initial examination little mention was made of associated lesions. Subsequent review and examination has revealed that all of the members except one have one or both of bony and soft tissue tumours. As in *Family 1* the epidermoid cyst was the lesion most frequently encountered. However, two of these five patients in particular warrant further mention.

Case 1.—R.T., a 36-year-old man, had an ileostomy in 1950 followed by a colectomy and ileorectal anastomosis. Approximately two years later he was readmitted because of "a hard round lump 1" in diameter present in the ileostomy scar". This was believed to represent metastatic tumour. At operation, the lesion was found lying in the rectus muscle and histological section showed a typical desmoid complete with muscle invasion (Fig. 6).

Case 2.—In 1952, this man, F.T., had a total colectomy with perineal resection of the rectum and ileostomy. Pathological examination revealed diffuse polyposis with multiple invasive carcinomata with lymphatic spread. Two years later when he was readmitted because of hydronephrosis, he complained of a fist-sized mass in his mid-abdomen. This was believed on clinical grounds to represent metastatic tumour. Laparotomy revealed "the entire small bowel mesentery was infiltrated by a grapefruit-sized hard mass growing between the leaves of the mesentery". Multiple biopsies were taken and showed only dense fibrous tissue. Four months later he was readmitted in uremia and died. Postmortem examination revealed this large mesenteric mass to be obstructing both ureters. Microscopical examination showed a central focus of tumour cells surrounded by thick layers of dense hyalinizing connective tissue, so that while carcinoma was present, there was a degree of fibroblastic proliferation in excess of normal tissue response which was responsible for the ureteral obstruction with its fatal outcome.

The involved members of this family have produced eight children ranging from 11 to 19 years. The eldest of these, a 19-year-old girl, just recently has been found on sigmoidoscopy to have multiple polyps, as well as osteomata on her skull and hard palate. The other children are currently being contacted for investigation.

Treatment

Removal of all adenomata is mandatory, either by colectomy with perineal excision of the rectum or colectomy with preservation of the rectal stump and ileorectal anastomosis.

If the latter course is chosen, not more than 15 cm. of rectal stump should be left, so as to facilitate follow-up endoscopic examination and fulguration. It has been shown that 5 cm. of rectum will maintain sphincter-plus-reservoir control.[7] An end-to-end anastomosis should be employed to avoid leaving a blind pouch of rectum to harbour adenomata, and the rectal mucosa near the anastomosis should be cleared of polyps to prevent turning any in at the suture line. Because of the great absorptive powers of the terminal ileum, postoperative diarrhea can be minimized by resection of as little as possible of this segment of gut.[8]

Above all, the co-operation of the patient is essential if the rectum is to be spared,

since endoscopic examination with fulguration of residual or new adenomata is necessary at intervals indefinitely. Despite periodic examination, this operation is not without risk, for malignant changes occur in polyps in the rectal stump in up to 20% of cases.

In *Family 1*, all six were subjected to colectomy and ileorectal anastomosis within the past year. The operation was preceded by one to three fulgurations of the rectal segment. There were no deaths or complications and all have an excellent functional result with one to four soft but formed stools daily.

Pathological examination revealed that two members of the family had polyps which showed intraepithelial malignant change, but there was no instance of invasive carcinoma.

Postoperative endoscopy has been carried out and residual adenomata fulgurated when necessary.

Family 2 was treated initially by several different surgeons between 1937 and 1952. Two of the family had frank carcinoma at the time they were first seen and both are dead at ages 27 and 38 respectively. One died of complications after an ileostomy before a colectomy could be performed; the other died in uremia as previously described.

The remaining three have had colectomies with ileorectal anastomosis and are well with satisfactory functional results. Two had a side-to-side anastomosis leaving a blind rectal pouch. In two instances polyps showing cellular atypism consistent with early malignant change were noted.

Discussion

A high incidence of bony and soft tissue lesions is apparent in these two groups with virtually every member of both families showing one or both of these additional manifestations.

This is probably commoner than generally recognized since presence of sebaceous cysts unless unusually large or numerous often fails to be recorded. Furthermore the osteomata are frequently so flat as to escape notice unless specifically looked for and in two patients were not palpable at all, being noted radiologically following skeletal survey.

While there is no evidence that sebaceous cysts or osteomata are of any serious clinical significance, it is of interest that two members of *Family 1* had multiple cysts removed many years before the diagnosis of polyposis. Thus investigation of the colon in anyone displaying multiple cysts or osteomata may lead to earlier recognition of a potentially lethal disease.

The tendency to excessive fibroblastic proliferation can, on the other hand, be of considerable significance and was undoubtedly a contributory factor in the mesenteric mass which produced the ureteral obstruction with its fatal outcome in a member of *Family 2*.

Colectomy with ileorectal anastomosis has proved to be a satisfactory method of treatment to date in all cases except one of the two members of *Family 2* who had a side-to-side anastomosis. This has left a blind pouch of rectum which has been so difficult to visualize and fulgurate subsequently that consideration is being given to re-operation with conversion to an end-to-end anastomosis.

The living members of *Family 2* were operated upon 14, 10 and 7 years ago respectively and re-check endoscopy with fulguration has been performed every six to 12 months with an occasional two to three-year interval. On each examination new crops of adenomata were noted, as many as 20 to 30 when the time lapse had been lengthy. It would seem therefore that the rectal mucosa retains its proliferative characteristics indefinitely and the need for continuing periodic re-check must be stressed.

The eldest and most extensively involved member of *Family 1* showed a considerable number of adenomata still remaining at his first postoperative check, about half of which were fulgurated. At subsequent examination six weeks later only one adenoma was found. This is the only instance in either group suggesting spontaneous regression of polypi after ileorectal anastomosis, a phenomenon which has been reported by a number of observers.[9, 10]

Summary and Conclusions

Eleven cases of multiple polyposis in two families are reported, every member of one family of six being involved. Invasive carcinoma was present in two. Patients with polyposis have a tendency to excessive proliferation of cells of mesenchymal origin. A high incidence of associated bony and soft tissue lesions is noted. Colon investigation in people with multiple cysts or osteomata may lead to earlier recognition of polyposis. Colectomy and ileorectal anastomosis is a satisfactory method of treatment. End-to-end anastomosis is indicated. The importance of re-check endoscopic examination over an indefinite period is stressed. Spontaneous regression of polyps following operation has not been a feature in this study.

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Résumé

La polypose multiple familiale du côlon a été décrite en 1882 par Harrison Cripps. On admet généralement maintenant que la maladie a une origine héréditaire (il s'agit d'une mutation génétique); elle est transmise de génération en génération selon les lois de Mendel, comme un facteur dominant, non lié au sexe. Pratiquement l'affection se transforme toujours en cancer, et ceci survient plus tôt que ce n'est le cas pour des carcinomes du gros intestin. Bien que le symptôme majeur soit une apparition d'un grand nombre de petits polypes intéressant toute la muqueuse des côlons, on rencontre fréquemment des tumeurs des tissus mous ou des os. L'auteur rapporte ici l'analyse de cette pathologie dans deux familles. Dans la première famille, la mère était morte d'une tumeur hépatique qui très vraisemblablement était une métastase d'un carcinome du côlon: les six enfants furent atteints de polypose et l'on trouva aussi un grand nombre de tumeurs des os et des tissus mous: ostéomes, fibromes. Dans la seconde famille cinq membres furent atteints sur huit, et deux d'entre eux développèrent un carcinome du gros intestin. Le traitement de cette maladie, forcément compliqué et variable selon les cas, est discuté en détail: la colectomie suivie d'anastomose iléo-rectale a donné des résultats satisfaisants. Ces cas doivent être surveillés de près pendant très longtemps. De plus il faut noter que la vérification du gros intestin doit être entreprise toutes les fois que l'on se trouve en présence d'un malade porteur de nombreuses tumeurs du tissu conjonctif ou des os.

SURGERY OF THE STOMACH AND DUODENUM. Edited by Henry N. Harkins and Lloyd M. Nyhus, with 43 contributors. Foreword by Sir Charles Illingworth. 736 pp. Illust. Little, Brown & Company, Boston; J. B. Lippincott Company, Montreal, 1962. $29.50.

This is an extremely comprehensive and critical review of the subject written by world-renowned authorities in this field. The authors-in-chief set out three important criteria for gastric surgery: (1) Surgical procedures must be based on a sound physiologic background. (2) Any resective operation must be applied quantitatively. (3) Operative therapy must be individualized to fit the needs of each patient.

These criteria are adhered to throughout the book and the presentation of each topic is supported by relevant data, individual comment and essential publications. This work will undoubtedly be acknowledged as the authoritative text on the surgery of this region. To the postgraduate student in surgery, the gastroenterologist and the practising surgeon, this book gives a more complete understanding of a difficult subject.

THE USE OF GASTROSTOMY IN SURGERY*

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GASTROSTOMY is not a new operation. When introduced over a century ago, its chief use was as a permanent opening into the stomach to provide a means of feeding patients with disease of the esophagus. More recently, gastrostomy has been used with increasing frequency on a temporary basis to provide postoperative decompression when it was desirable to avoid the use of a nasogastric tube. One hundred and four gastrostomies performed at the University of Alberta Hospital during the last six years, 1956 to 1961 inclusive, were reviewed in an attempt to evaluate the procedure and sharpen our indications for its use.

PERMANENT GASTROSTOMY

TABLE I.—PERMANENT GASTROSTOMY

Stricture of the esophagus	5
Carcinoma of the esophagus	5
Perforation of the esophagus	3
Fistula (tracheoesophageal)	2
Carcinoma of the mouth	2
Burns of the mouth	1
Total	18

Eighteen gastrostomies were of the permanent variety, in that they were created to provide for long-term feeding of the patient (Table I). In five patients with esophageal stricture, the gastrostomy opening was also used for retrograde dilatation of the esophagus.

In 1837 Egeberg suggested gastrostomy for carcinoma of the esophagus in an attempt to alleviate malnutrition. Witzel protected the gastric opening from leak by creating a tunnel of stomach wall around the tube. In 1913, Janeway formed a flap of the anterior wall of the stomach and brought it out as a fistula, thus avoiding internal leakage, but at the same time causing considerable irritation of the skin of the abdominal wall by gastric juices. One-third of the 18 gastrostomies in this group were of the Witzel type, while the remainder were of the Stamm variety in which a Foley catheter was inserted through the stomach wall and held by double purse-string sutures.

In patients with carcinoma of the esophagus, there is no indication that gastrostomy has prolonged life or has significantly increased the comfort of the patient during the terminal phases of the disease. The five patients with permanent gastrostomy for carcinoma of the esophagus were all seen early in our series. More recently, carcinoma of the esophagus has been treated by a direct attack on the lesion, or by short-circuit procedures which have proved more satisfactory than gastrostomies for palliative purposes. In patients with benign lesions of the esophagus, problems of malnutrition have been markedly improved by gastrostomy, often making major surgical procedures possible.

TEMPORARY GASTROSTOMY

TABLE II.—TEMPORARY GASTROSTOMY

Peptic ulcer	42
Intestinal lesions	23
Hiatus hernia	7
Miscellaneous	14
Total	86

Eighty-six gastrostomies were of the temporary variety performed as adjuncts to other surgical procedures for purposes of providing gastrointestinal decompression (Table II). As might be expected, most temporary gastrostomies were performed in association with operations on the gastrointestinal tract where it was de-

TABLE III.—TEMPORARY GASTROSTOMY WITH PEPTIC ULCER OPERATIONS

Vagotomy and pyloroplasty	24
Vagotomy and gastroenterostomy or hemigastrectomy	9
Subtotal (75%) gastrectomy	8
Vagotomy alone	1
Total	42

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sirable to protect the suture line from undue distension, and of these, operations for peptic ulcer comprised almost 50% of the series.

Forty-two temporary gastrostomies were due distension, and, of these, operations for complications of peptic ulcer (Table III). Early in the series, one vagotomy alone was done. Vagotomy alone is now very rarely performed as it requires some form of drainage of the stomach to avoid the unpleasant complications associated with gastric retention. The 75% resection of the stomach, particularly of the Billroth II type, usually empties readily, and many institutions have abandoned any form of decompression as a routine measure in the postoperative period.

The recent trend to more conservative resections protected by a vagotomy has overcome many of the nutritional problems associated with the 75% gastric resection. However, a vagotomy and hemigastrectomy of the Billroth I type almost always requires gastrointestinal decompression. In vagotomy and pyloroplasty, gastrointestinal function is even slower in returning to normal. Vagotomy and pyloroplasty combined with local suture is now considered by many to be the procedure of choice in elderly patients undergoing surgical treatment for active bleeding from a duodenal ulcer. In our series, gastrostomy was found to be ideally suited to vagotomy and pyloroplasty, as it prevents stasis and stimulation of gastric secretion, which prevent healing of the ulcer.

In most patients in this group, gastrostomy was utilized for gastric decompression postoperatively in preference to a nasogastric tube. The average age of these patients was 48 years (youngest 30 years, oldest 80 years), and the gastrostomy tube was kept in place for an average of eight days.

Technique of Temporary Gastrostomy

The technique of temporary gastrostomy basically involves the insertion of a No. 16 Foley catheter into the stomach through a stab wound in a relatively avascular area, midway between the greater and lesser curvatures; the catheter is secured by double purse-string catgut sutures. Suturing the serosa of the stomach to the parietal peritoneum decreases the possibility of leakage. Anchoring the stomach to the fascia of the abdominal wall as well as to the peritoneum may cause traction discomfort. Suturing the stomach to the peritoneum only is preferable, as some degree of flexibility is permitted. With a high resection of the stomach, the gastrostomy opening does not come into direct contact with the abdominal wall. In such a case, the tube can either be threaded through the omentum for protection against leak, or it can be passed extraperitoneally under the abdominal wall to its point of exit. Only 7 to 10 c.c. of fluid are required in the Foley bag. A larger balloon may stimulate gastric activity and may cause mechanical obstruction at the pylorus.

TABLE IV.—Temporary Gastrostomy with Intestinal Lesions

Small bowel obstruction	11
Colon resections	10
Volvulus of the sigmoid	1
Diverticulitis	1
Total	23

Seven patients in this series had temporary gastrostomies performed in conjunction with hiatus hernia repairs via the abdominal approach. In these patients, the gastrostomy tube was actually used as an ancillary feature of the operative repair of the hiatus hernia. If the tube is correctly positioned, it anchors the stomach to the anterior abdominal wall under sufficient tension, thereby helping to prevent recurrence of the hiatus hernia in the early postoperative period. At the same time, gastrostomy often eliminates the use of a nasogastric tube over a prolonged interval of time.

In this group of 23 patients undergoing bowel surgery, postoperative decompression was maintained by gastrostomy (Table IV). Generally the patients were considered to be poor operative risks because of age, cardiac status or general debility. The average age in this group was 67 years, and the tube was left *in situ* until the ninth postoperative day.

In this miscellaneous group of patients, gastrostomy was utilized for specific reasons (Table V). In all of the patients ex-

TABLE V.—Temporary Gastrostomy with Miscellaneous Procedures

Biliary tract	8
Carcinoma of the stomach	3
Carcinoma of the urinary bladder (ileal loop)	2
Acute pancreatitis	1
Total	14

cept one, bowel surgery was involved, so that gastrointestinal tract decompression was desirable. Commonly, the presence of chronic pulmonary disease or poor cardiac reserve did not permit major surgical procedures to be undertaken without maximum precautionary measures. Thus, gastrostomy was used to eliminate the potential hazards of nasogastric intubation.

All of the patients undergoing biliary tract surgery in this group were jaundiced. In four patients cholecystojejunostomies were performed for malignant obstruction in the head of the pancreas, and in the other four the duodenum was opened to permit a direct exposure of the ampulla of Vater. In patients with carcinoma of the stomach, the lesions were inoperable and gastrostomy was performed in conjunction with palliative procedures. In patients with carcinoma of the urinary bladder, ileal loop conduits were created.

With reference to complications of gastrostomy, Senter[1] reports a series of 50 gastrostomies in which three fatalities occurred, one as a result of leak producing a generalized peritonitis. In our series we were fortunate to have no deaths, and no generalized peritonitis or abscess formation that could be attributed to gastrostomy leakage (Table VI). Two elderly debilitated patients in our series eviscerated postoperatively. All of our gastrostomies were brought out through a separate stab wound rather than the main incision, and in both of these patients, the stomach remained attached to the abdominal wall without evidence of leak.

In two patients, severe hemorrhage occurred from the gastrostomy site in the stomach. One of these hemorrhages occurred early in the postoperative period; the patient was treated conservatively and responded to transfusion. This was unquestionably an error in technique. In the other patient, the bleeding was excessive

TABLE VI.—Complications of Gastrostomy

Leak-peritonitis-abscess	0
Bleeding	2
Persistent fistulas	2
Wound infection	2
Total	6

and re-exploration was required on the seventh postoperative day. Meticulous hemostasis at the time of the gastrostomy is mandatory, although mechanical irritation of the tube may be a precipitating factor in delayed hemorrhage. Delayed hemorrhage may be minimized by deflating the Foley balloon on the fourth day, thus decreasing the possibility of pressure necrosis of gastric mucosa.

A persistent fistula occurred in two patients. It is very common for a gastrostomy opening to discharge for two or three days following removal of the tube. Most of these close spontaneously with no special attention. In our experience, a routine of withholding food several hours before and several hours after removal of the gastrostomy tube has greatly facilitated the spontaneous closure of a gastrostomy wound. In the two persistent fistulas requiring surgical closure, the gastrostomy tubes were of large calibre, and both tubes were in place for over a month. During the last year we have made a point of bringing the gastrostomy tubes obliquely through the abdominal wall. This appears to have eliminated serious fistula formation.

All of the gastrostomy openings showed a slight inflammatory reaction externally owing to mechanical irritation of the tube. However, severe infections which required active treatment and prolonged the patient's convalescence period occurred in only two patients.

Discussion

Nasogastric tubes are poorly tolerated by infants and young children. Acute pharyngitis, abscess formation and otitis media commonly occur in this age group. Holder and Gross[2] have stated that a more liberal use of temporary gastrostomy has been one of the most significant single improvements made in recent years in the general care of young surgical patients.

It is well known that use of nasogastric tubes in elderly patients is associated with an increased incidence of postoperative pulmonary complications, especially if there is pre-existing pulmonary embarrassment. Moore[3] has shown that in severely ill surgical patients, the energy requirements for muscular activity, especially of the diaphragm and intercostal muscles, is not met. Bronchopneumonia then develops owing to failure of the host's respiratory muscles rather than invasion of the host. Therefore, any sparing of already precarious pulmonary function may make the difference between success and failure.

We have found by pulmonary function studies that after any abdominal surgery there is a decrease in volume exchange and rate of air flow into the lungs, which is compounded by co-existent lung disease and excessive pharyngeal secretions. Removal of the nasogastric tube postoperatively has resulted in immediate improvement in volume exchange and rate of pulmonary air flow. The use of intermittent positive pressure breathing and nebulization has greatly reduced our incidence of postoperative pulmonary complications. The efficiency of these methods is greatly reduced by the mechanical presence of a nasogastric tube.

Generally speaking, complications of gastrostomy most frequently occur because of errors in the technique used in its construction. When this procedure is performed properly, relevant complications are infrequent and are usually of little significance.

Summary

This review is concerned with all gastrostomies performed at the University of Alberta Hospital from 1956 to 1961 inclusive. The 104 gastrostomies carried out during this six-year period are divided into two groups. 1. Permanent gastrostomies performed as primary procedures. 2. Temporary gastrostomies done in association with other abdominal surgery.

Basically, permanent gastrostomies were utilized for feeding. Those done in association with other abdominal surgery served to alleviate the hazards, complications and discomfort of a nasal Levin tube postoperatively.

From this study, it becomes apparent that the need for permanent gastrostomy has largely disappeared. On the other hand, temporary gastrostomy performed in association with other abdominal surgery has proved to be of increasing value, and can be performed with reasonable safety. This procedure also has a particular place as an adjunct in hiatus hernia repair.

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Résumé

La gastrostomie est une intervention qui fut introduite dans la pratique chirurgicale voici environ un siècle. Son but est de créer une ouverture dans l'estomac par laquelle il sera possible de nourrir un malade souffrant d'une sténose œsophagique. Plus récemment on a appliqué cette technique à d'autres fins; à titre temporaire, on l'emploie dans des cas où une aspiration décompressive est nécessaire et ne peut être faite par tube naso-gastrique. Les auteurs présentent ici une étude statistique portant sur cent quatre gastrostomies effectuées à l'Hôpital de l'Université d'Alberta entre 1956 et 1961. Sur ce total, 18 furent faites de façon définitive pour les affections suivantes: rétrécissement de l'œsophage, cancer et perforation de l'œsophage, fistule trachéo-œsophagienne, carcinome ou brûlures de la bouche. Une série de 86 autres interventions furent effectuées à titre temporaire, dans le but de provoquer une décompression du tratus gastrointestinal. Bien entendu ces opérations étaient associées à d'autres procédés opératoires; on trouve dans ce groupe: des ulcères peptiques, des lésions intestinales, des hernies du hiatus de Winslow. Enfin 42 avaient pour but de lutter contre des complications d'ulcère peptique. Elles furent faites en conjonction ou à la suite des interventions suivantes; vagotomie, pyloroplastie, gastroentéroanastomose, gastrectomie subtotale. La technique de la gastrostomie temporaire est décrite. On introduit dans l'estomac, dans une région autant que possible pauvre en vaisseaux, un cathéter de Foley, à mi-chemin entre la grande et la petite courbure. Ce cathéter est fixé par une suture en bourse au catgut. Il semble inutile de fixer l'estomac à la paroi abdominale antérieure, une suture amarrant le péritoine pariétal est suffisante. En règle générale, lorsqu'on retire le tube de Foley, la bouche de gastrostomie a une tendance naturelle à se refermer. Cependant, dans deux cas il y eut formation d'une fistule gastrique qui nécessitèrent une suture chirurgicale.

HEREDITARY MULTIPLE DIAPHYSEAL SCLEROSIS: RIBBING'S DISEASE OR ENGELMANN'S DISEASE?*

J. C. FAVREAU, M.D., V. SISON, M.D., P. LABELLE, M.D. and CARROLL A. LAURIN, M.D., *Montreal*

In 1949, Ribbing[12] described a skeletal condition affecting four members of one family; the condition has since been referred to as Ribbing's disease or hereditary multiple diaphyseal sclerosis. Since the original description, only one report has been published, namely, Paul's article[11] in 1953. However, Paul later rectified his diagnosis when a sibling of his patient developed the complete clinical picture of Engelmann's disease.

The scarcity of case reports describing Ribbing's disease is difficult to account for; two possible explanations come to mind: either it is in fact a very rare condition, or it is not a specific entity but rather a variant of Engelmann's disease. The latter view is held by Lennon, Schechter and Hornabrook,[8] who have conveniently grouped all cases of familial metaphyseal sclerosis under one entity, namely, Engelmann's disease. Their suggestion has obvious didactic advantages and, on that basis, has merit. However, the clinical picture of Ribbing's disease and that of Engelmann's disease are so dissimilar that, in Griffiths' opinion,[5] it is more accurate to distinguish them and to maintain separate diagnoses. Admittedly, the two conditions can be indistinguishable histologically and radiologically, but their prognosis and their clinical picture are so different that it does not suffice to explain that they are mild and severe forms of a poorly understood disease. Engelmann's disease and Ribbing's disease may eventually prove to be associated conditions; but, until more is known about both lesions, the authors feel that it is more desirable to consider them and to report them separately.

Case Report

C.T., a 5-year-old girl, was first seen on November 30, 1960, when she complained of pain in the left tibia. The pain had appeared 15 days earlier and was described as dull and boring in nature. There was no history of an accident or of any recent illness. Examination revealed a warm fusiform swelling over the anterior aspect of the left tibia. Except for a birthmark at the base of the neck (Fig. 4b), the physical examination was normal. A radiograph of the left tibia (Fig. 1a) revealed a dense periosteal reaction with thickening and sclerosis of the diaphyseal cortex. A tentative diagnosis of osteomyelitis was made and antibiotics were given empirically for a period of two weeks. One month later, the tenderness and warmth had subsided but the swelling persisted. Further radiographs (Fig. 1b) suggested slight regression of the disease process (on retrospect, the authors now feel that the suggestion of improvement was entirely due to a difference in x-ray techniques). Two months later, the patient complained of slight pains in the right leg. The right tibia now showed a similar swelling with slight tenderness on palpation. Radiographs (Fig. 2a) revealed a comparable image of dense periosteal reaction with diaphyseal sclerosis. Again, antibiotics were administered for two weeks, and the clinical signs and symptoms subsided with definite radiographic evidence of improvement two months later (Fig. 2b).

On June 27, 1961, seven months after the original visit, the signs and symptoms recurred on the left side, the site of the original involvement. Again physical examination revealed a fusiform swelling over the left tibial crest measuring approximately 3" in length. The swelling was hard, had a smooth surface and was warm and tender to touch. The right leg was now asymptomatic. The physical examination was otherwise normal. Laboratory examination included a complete blood count, sedimentation rate, serology, urinalysis, serum phosphatase, serum calcium, total proteins and blood culture; all were normal. Radiographs revealed a progression of the diaphyseal sclerosis with a partial obliteration of the intramedullary canal on the left side (Fig. 1c). A skeletal survey revealed periosteal changes and cortical thickening in the left ulna (Fig. 3a). The patient had never complained of forearm pains.

A biopsy of the left tibia was performed eight months after the onset of symptoms (Fig. 1d). The bone was relatively avascular

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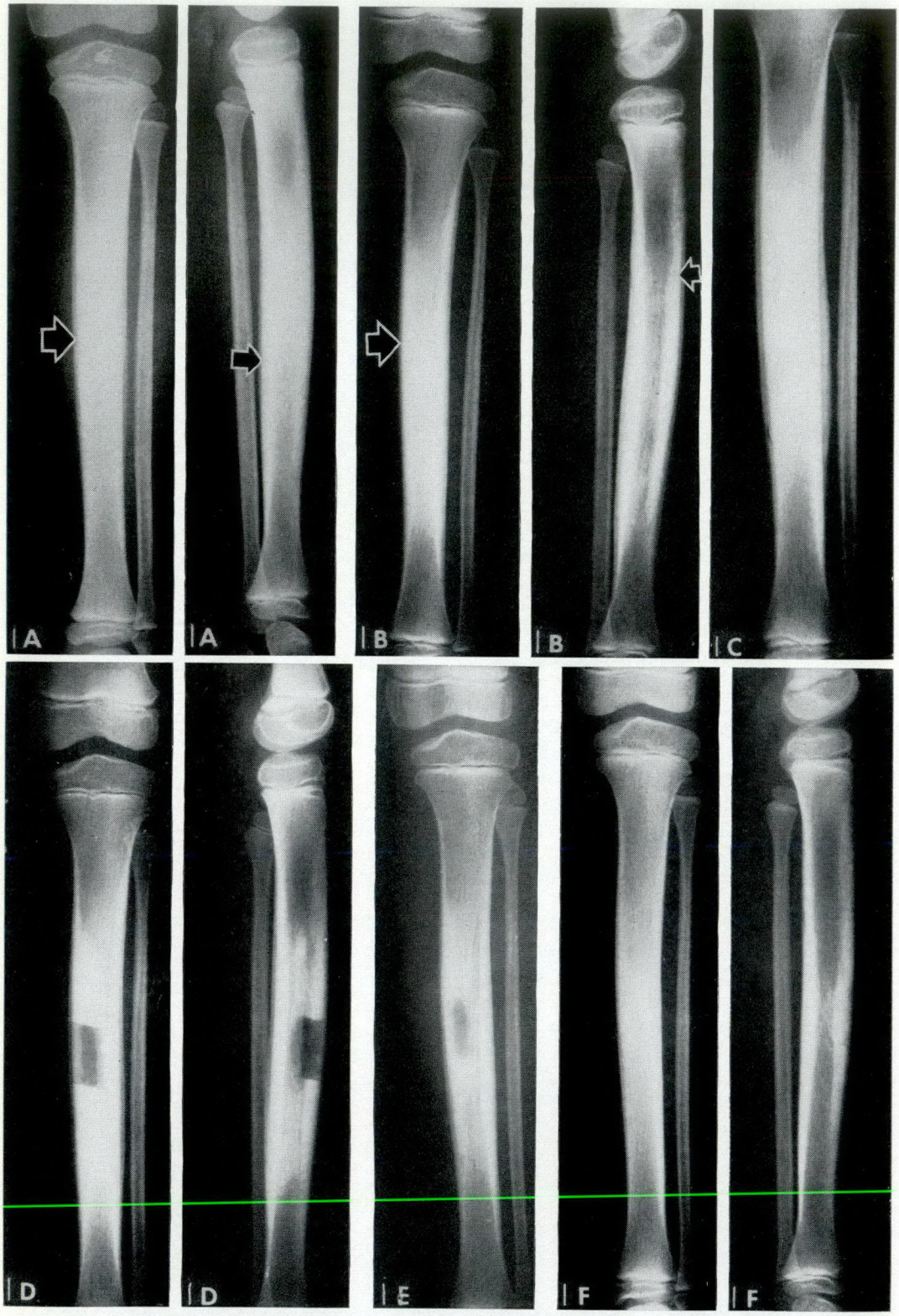


Fig. 1.—Left leg: Serial radiographs demonstrating (a) (b) and (c) the progression of the disease process; (d) the site of biopsy; (e) and (f) the subsequent gradual regression of the disease process (see text for clinical details).

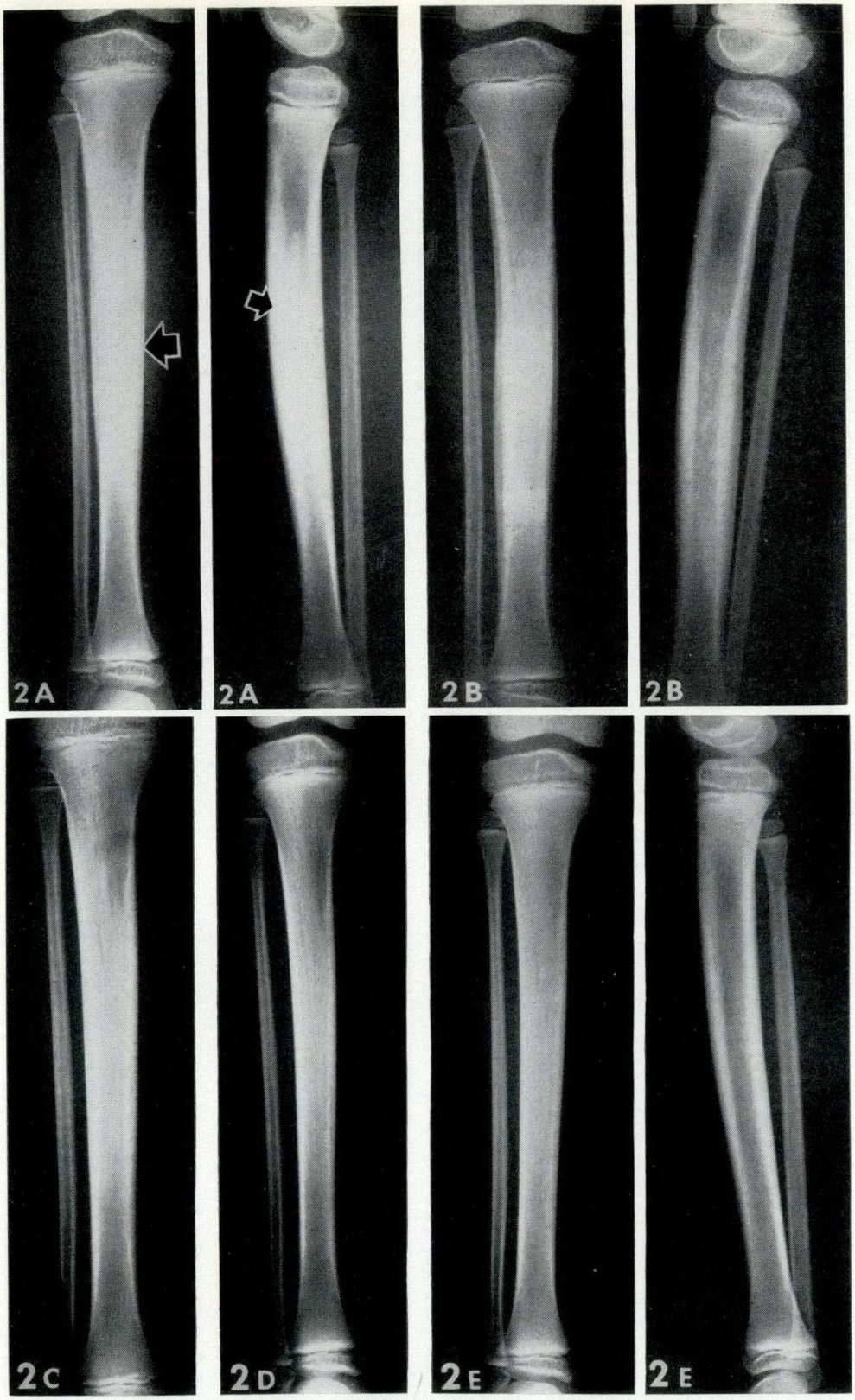


Fig. 2.—Right leg: Serial radiographs showing the gradual regression of the disease process. Note that the last radiograph on the right leg (e) shows more complete regression when compared with the left leg (Fig. 1f). Indeed, the regression of the disease process in the right tibia was already apparent in April 1961 (b), while it did not begin on the left side until July 1961 (Figs. 1c and d). In other words, the disease process runs a similar course in different limbs but not necessarily a simultaneous one (compare with Figs. 3a, b and c).

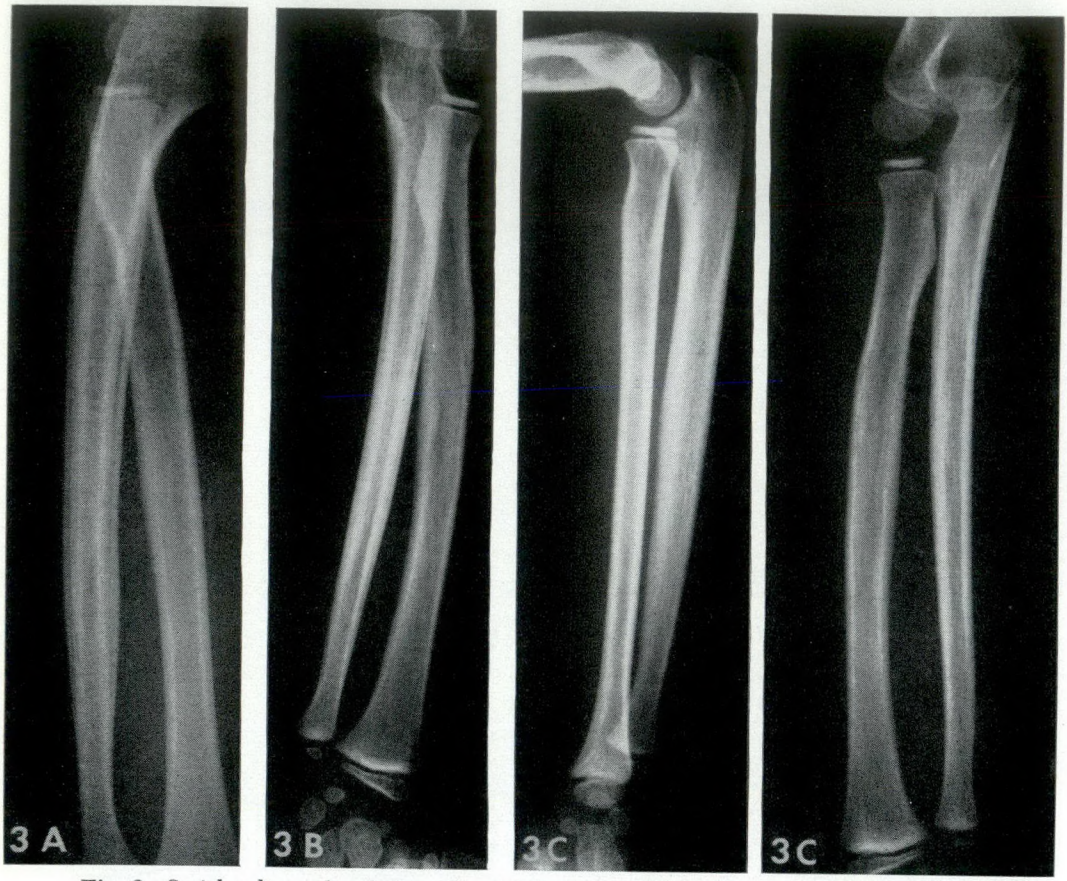


Fig. 3.—Serial radiographs of the patient's left forearm illustrating the regression of the disease process.

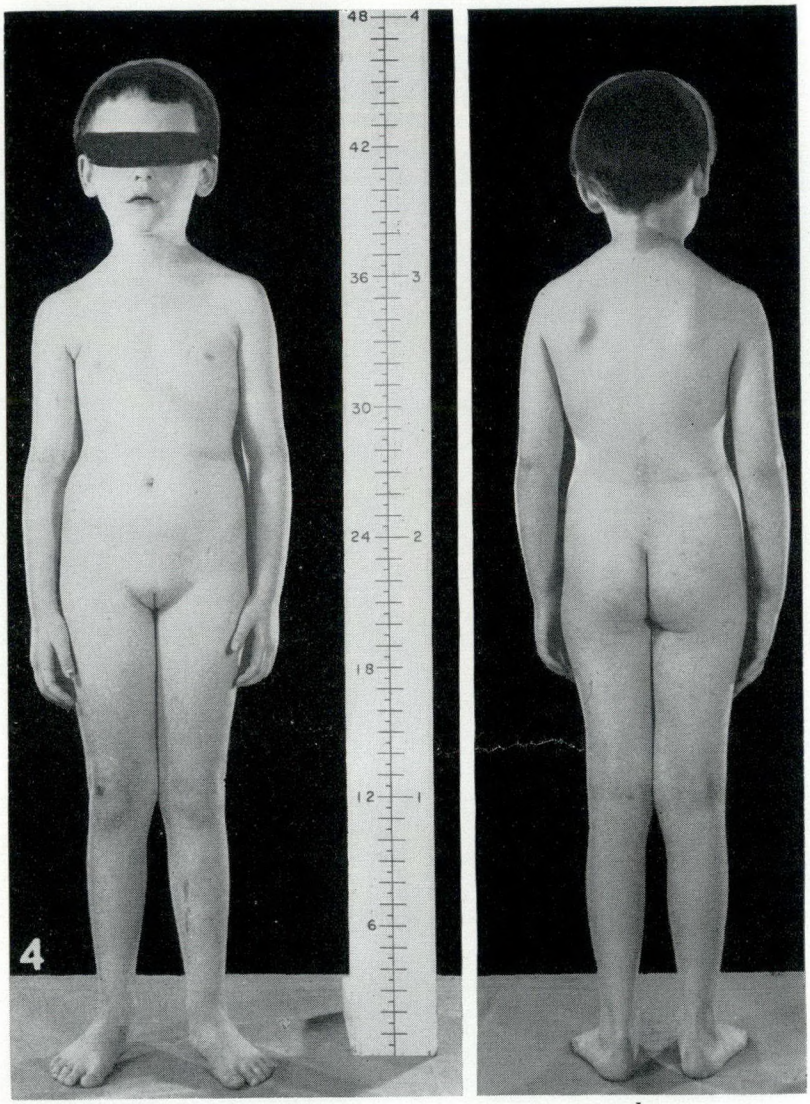
and hard; the cortex was excessively thick with complete obliteration of the intramedullary canal. The histological examination (Figs. 5a and b) revealed areas of marked osteoblastic activity with wide, irregular seams of incompletely calcified new bone (Fig. 5a); other more mature areas (Fig. 5b) revealed an increased number of otherwise normal osteoblasts imbedded in unusually wide bony trabeculae. There were no signs of inflammatory reaction. A culture of this bone was negative. Without further treatment, the disease process became clinically quiescent and radiographs confirmed a gradual regression on both sides (Figs. 1e and f; 2c, d and e). The radiographic changes noted in the left ulna also subsided (Figs. 3b and c).

Family History

Although a chromosomal analysis of this patient revealed no anomaly, an investigation of the child's relatives uncovered a familial incidence on the maternal side. None had ever previously complained of pertinent symptoms, yet the child's mother, two sisters and a maternal cousin were noted to have a definite periosteal reaction of one or more bones (Figs. 6a, b, c and d; Figs. 7a, b, c and d). The radiographic signs noted in four of her immediate relatives are admittedly not gross, nor are they impressive, but they have been verified by experienced radiologists as being definitely abnormal.

Discussion

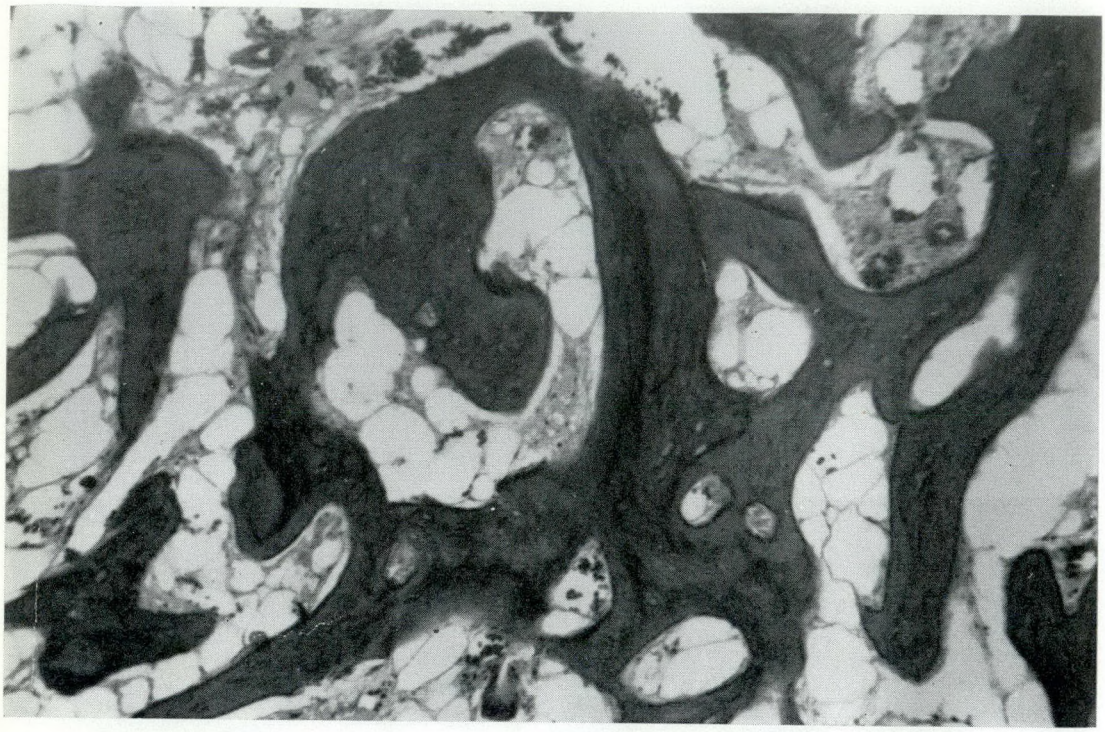
The concept of Engelmann's disease has evolved considerably since its original description by Camurati[2] who, incidentally, described the condition eight years before Engelmann.[3] None the less, its cause and pathogenesis are still unknown. Authors such as Battaglia and Venturi,[1] Fairbank,[4]



a b

Fig. 4.—Clinical photographs of the patient: normal body configuration. Note (a) the biopsy scar on the left leg, and (b) the birthmark at the base of the neck.

Holt,[6] Jackson *et al.*,[7] Mikity and Jacobson[9] and Neuhauser *et al.*[10] had insisted previously that the Engelmann's disease was infantile, symmetrical, progressive and non-familial. Nevertheless, the condition is now considered to be hereditary; it need not be symmetrical nor progressive, and it may become clinically apparent at any age, even during adulthood. It is considered to be a systemic disease, with clinical manifestations which spread far beyond the osseous system. The mental, neurological and endocrine manifestations of Engelmann's disease have been well described and are usually present in varying degrees of severity. The extremities of such patients frequently appear unduly long, and this anomaly is usually related to hormonal factors. Finally, the bone pathology is progressive for an undetermined period of time and eventually becomes stationary; although some cases may have an asymmetrical evolution, the final radiographic appearance is almost always symmetrical.



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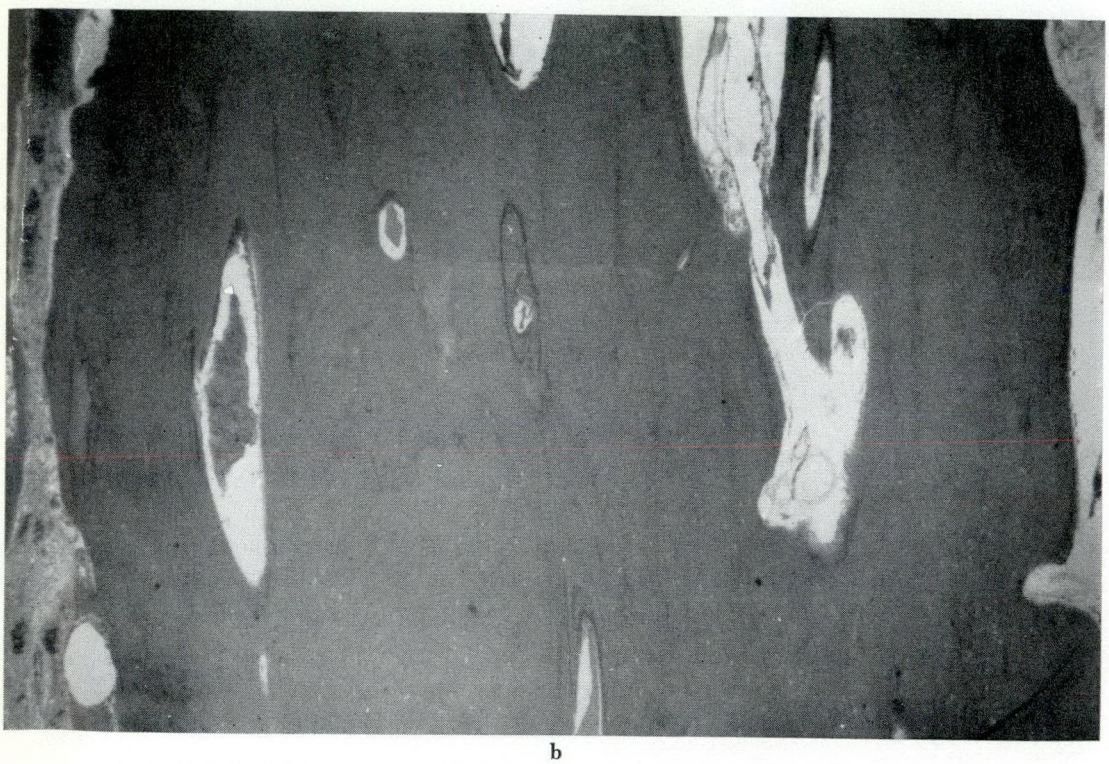

b

Fig. 5.—Histological appearance of the biopsy specimen (see text); (a) and (b) represent different fields.

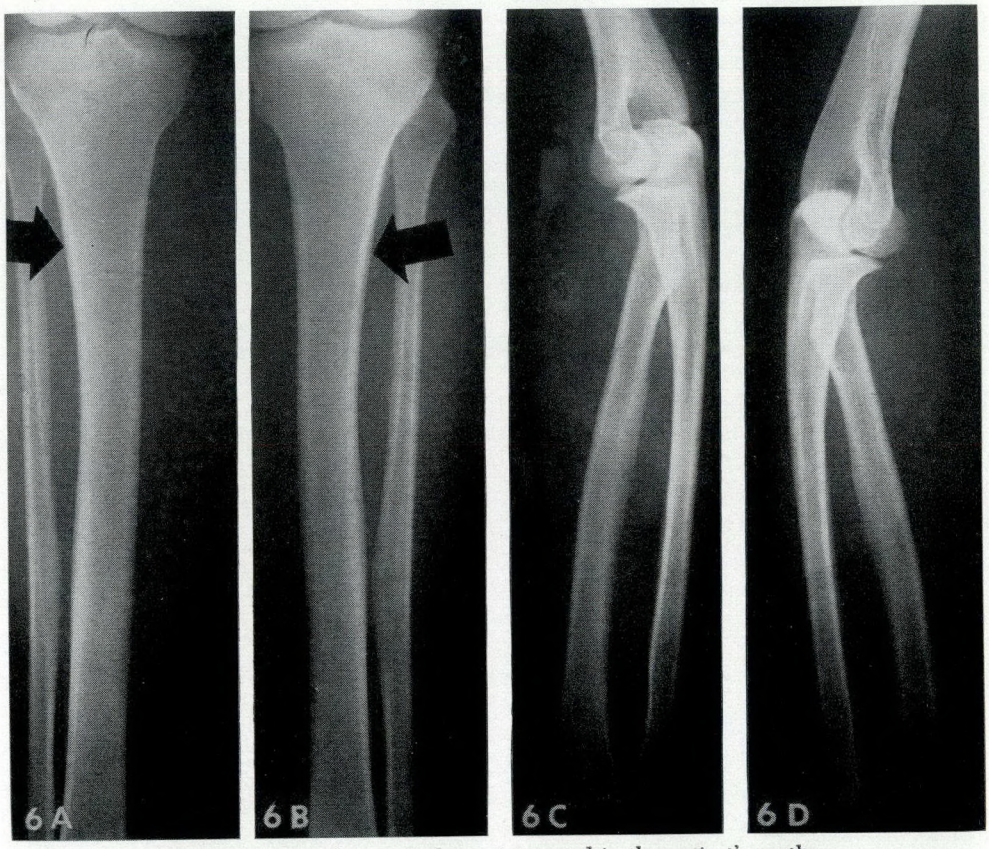

Fig. 6.—Abnormal periosteal reaction noted in the patient's mother.

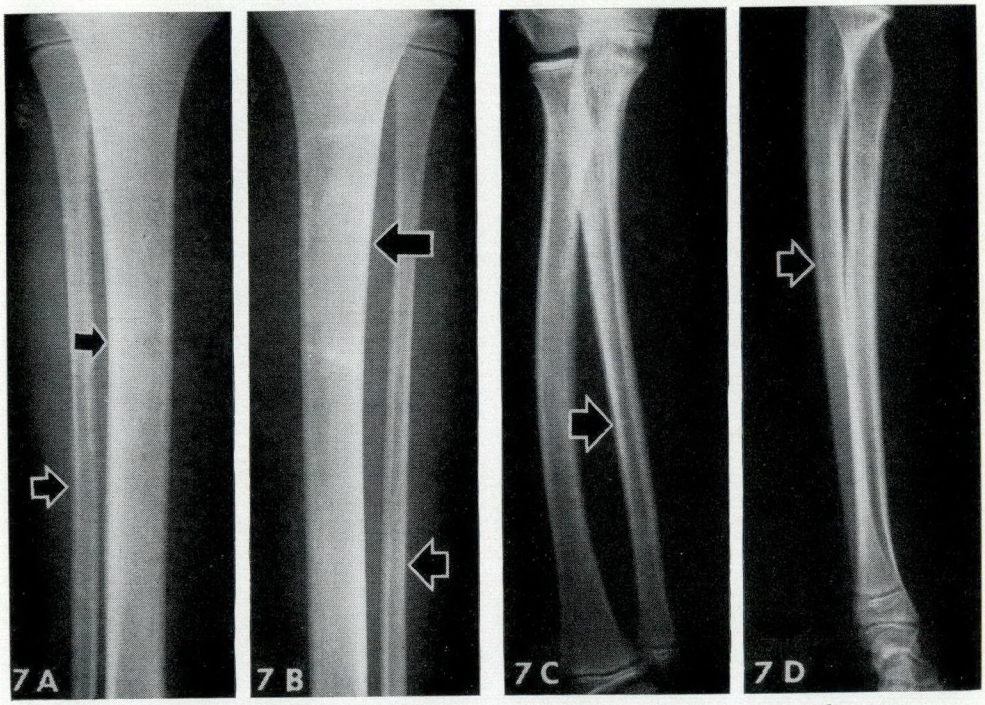

Fig. 7.—Periosteal changes noted in the tibiae and forearm of two maternal cousins.

Lennon, Schechter and Hornabrook[8] are probably correct when they suggest that the clinical picture of Engelmann's disease can vary considerably; indeed, it may be more accurate to refer to Engelmann's syndrome rather than to Engelmann's disease.

Are this child and her relatives mild and atypical examples of Engelmann's disease? The authors do not think so. These cases are true examples of Ribbing's original description. The possibility that the child described here may later develop neurological or endocrine signs, and hence be an example of Engelmann's disease, appears remote because none of her adult relatives have ever had such symptoms. Indeed Ribbing's disease appears to be entirely restricted to the osseous system. It is worth stressing that this patient is the first reported case where the radiological changes have shown definite regression. Ribbing suggested that the condition of one of his patients had regressed, but he did not illustrate it radiologically. This peculiarity, namely, the radiological regression of the pathological process is probably the main reason why this condition should be distinguished from Engelmann's disease. Although Engelmann's disease may become stationary, it has never been shown to regress. The excellent prognosis and the strictly osseous manifestations of Ribbing's disease are further reasons for recognizing this condition as a distinct entity.

Summary

A case of Ribbing's disease or hereditary multiple diaphyseal sclerosis is reported. A progression and a regression of the disease process has been demonstrated. The pathological process appears to be limited to the skeleton and may occasion no systemic manifestations; indeed the patients may be entirely asymptomatic and may belong to any age group. The prognosis is excellent. The authors suggest that Ribbing's disease and Engelmann's disease should be considered as separate entities.

The authors would like to express their sincere appreciation to the Medical Arts and Photography Department of the Ste-Justine Hospital, Montreal.

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Résumé

Histoire d'un cas. Une petite fille de cinq ans est vue pour la première fois en novembre 1960 pour des douleurs du tibia gauche. Il n'y a aucun contexte traumatique. A l'examen, on constate l'existence d'une tuméfaction fusiforme de la face antérieure de la jambe et la radiologie montre une réaction périostique de densification avec un épaississement et une sclérose du cortex diaphysaire. Pour éliminer le diagnostic d'ostéomyélite, on donne, à titre d'essai, un traitement aux antibiotiques pendant deux semaines. Deux mois plus tard, l'image radiologique était sans changement. Par ailleurs, l'examen général ne révélait rien d'anormal. On pratiqua alors une biopsie; le cortex du tibia était très épais. Au microscope on nota une abondance d'ostéoblastes mais aucun signe d'inflammation. Il s'agissait d'une maladie d'Engelmann. L'étiologie de ce syndrome est inconnue, mais la plupart des auteurs considèrent actuellement qu'il s'agit là d'une affection héréditaire. La maladie suit d'abord un cours ascendant, attaquant progressivement les diaphyses de divers os, symétriquement ou non, puis elle se stabilise. Dans le cas présenté ici, le diagnostic differentiel entre une maladie d'Engelmann et une maladie de Ribbing est discuté.

ENDOSCOPIC SURGERY OF PHARYNGEAL DIVERTICULA (Dohlman Technique): A Report of Two Cases*

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IN THIS report, two cases of pharyngeal diverticulum managed endoscopically by the Dohlman technique are presented and can be added to the five cases reported by Dohlman and Mattsson in 1960[2] and the 11 reported by Holinger and Johnston in 1961.[3] During the 20-year period in which this technique was developed, Dohlman[6] reports that he has operated on more than 100 cases with no serious complications and with highly satisfactory results.

In a cinéroentgenological study of the swallowing mechanism, Dohlman and Mattsson[1] illustrated in a convincing way the development of a Zenker's diverticulum. In normal swallowing, the larynx moves anterosuperiorly, lifting the arytenoids away from the posterior pharyngeal wall, and opening the lumen of the esophagus. The posterior portion of the cricopharyngeal muscle forms the posterior pharyngeal ring which is attached to the prevertebral fascia by areolar tissue. This areolar tissue permits a free sliding movement of the pharyngeal ring on the prevertebral fascia, but normally resists separation of pharynx from prevertebral fascia by traction force. Thus, in swallowing, when the larynx moves anterosuperiorly, the lumen of the esophagus is opened, and the stretch-receptors in the cricopharyngeus muscle are stimulated to initiate the reflex relaxation of the cricopharyngeus muscle (parasympathetic) preceding the passage of a food bolus.

In the ageing process, the prevertebral areolar tissue weakens and permits the pharynx to be pulled away from the prevertebral fascia. In swallowing, the movement of the larynx now lifts the whole pharynx up and forward because it is not firmly anchored posteriorly. The lumen of hypopharynx and esophagus fails to open normally, and the stretch-receptors of cricopharyngeus are not stimulated to initiate relaxation of the sphincter. Thus the stage is set for abnormal intraluminal pressures to cause herniation of the esophageal mucosa posteriorly through the weak triangle between the oblique and circular (cricopharyngeal) fibres of the inferior pharyngeal constrictor. As the hernial sac enlarges downwards into the superior mediastinum, it comes to occupy a dependent position, with the esophageal lumen lying in a transverse plane anteriorly. The anterior lip of the sac is formed by the posterior portion of the cricopharyngeus, and hence the act of swallowing actually closes the esophagus and opens the orifice of the diverticulum.

As a result of the diverticulum, the patient suffers dysphagia, regurgitation, malnutrition and inanition. Complications arising include recurrent aspiration pneumonia, chronic bronchitis, and occasional pulmonary abscess. Foreign bodies may impact in the diverticulum and subsequently cause perforation leading to mediastinitis. Localized esophagitis in the sac may occur, and foul odour from bacterial putrefaction of contents has been reported.

The medical management of esophageal diverticula is not rewarding. Before antibiotics, the surgical treatment was fraught with the dangers of mediastinitis. Consequently, the two-stage operation perfected by Lahey and Warren[4] was employed; the sac was dissected free and suspended to the cervical fascia with the ostium dependent. This permitted fibrosis to seal it off so that at the second stage the sac could be excised without contaminating the mediastinum. Subsequent to the development of antibiotics, a one-stage excision of the diverticulum could be carried out with relatively little risk of mediastinitis.

Complications arising in a series of 425 cases reported by Boyd[5] included recurrence in 13, recurrent nerve injury in 12, prolonged fistulas in three, severe stricture in three, and single cases of mediastinitis and pulmonary abscess. There were two operative deaths. It must be added that in this large series most of these complications did not occur in recent years.

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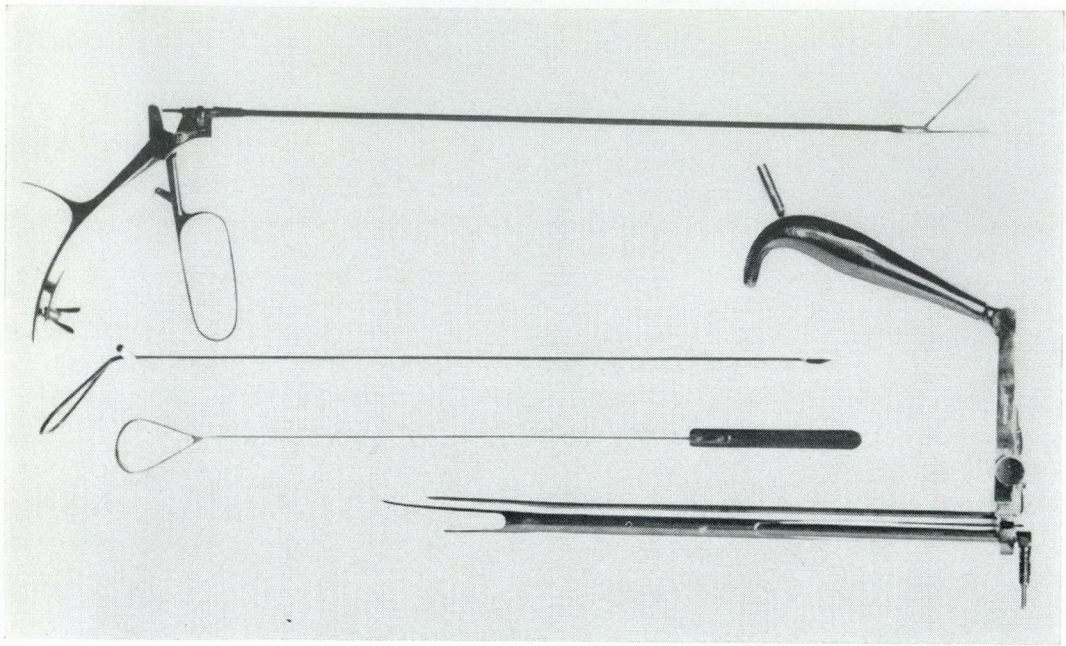

Fig. 1.—Instruments for the Dohlman procedure.—Top to bottom: (a) Grasping forceps for initial application of coagulation current. (b) Insulated knife for final division of cricopharyngeus using cutting current. (c) Insulated spatula for protection of posterior wall of sac during use of the knife. (d) The Dohlman-type scope; bivalved at the distal end, and with a post on the handle for connection to the ground lead on diathermy unit.

In the authors' experience, a satisfactory surgical repair of large diverticula is often difficult because of the size of the sac and the narrowness of the esophageal opening, so that persisting cricopharyngeal dysfunction gives rise to persisting dysphagia of some degree and to the possibility of recurrence. This is emphasized by the recent suggestion of musculoplastic procedures on the cricopharyngeus at the time of operation. The Dohlman technique, by dealing primarily (and only) with the cricopharyngeus, solves the problem of persisting cricopharyngeal dysfunction.

The technique of endoscopic surgery for cricopharyngeal diverticula, originally described by Dohlman and Mattsson[2] and later by Holinger and Johnston,[3] is carried out under general endotracheal anesthesia employing muscle relaxants. The duration of anesthesia usually need be no longer than 15 or 20 minutes. The specially designed scope is bivalved at its distal end (Fig. 1) and is positioned with the anterior lip in the lumen of the esophagus and the posterior lip in the ostium of the sac. This presents the cricopharyngeus muscle to the surgeon as a transverse ridge in the lumen of the esophagoscope. Insulated alligator forceps, the jaws of which extend down the dividing ridge for a distance of about one inch, are used to grasp the cricopharyngeus in the midline. Coagulation cautery current is applied to a pole on the forceps, using the Dohlman scope itself as ground. The current is applied until the muscle is visibly cauterized, and then the forceps are removed. The insulated spatula is then inserted into the sac of the diverticulum, and under direct vision the coagulated portion of the cricopharyngeus is divided with a small insulated knife, cutting against the spatula, and employing a cutting cautery current. Thus, the cricopharyngeus is completely divided in the midline posteriorly. Bleeding is minimal or absent. A small wedge of Gelfoam may be inserted between the divided edges of the muscle.

Postoperatively, the patients were maintained on intravenous fluids only for about 36 hours. It is not uncommon for the physician to find that such a patient develops a low-grade fever without chest pain or emphysema in the first 24 hours, but this

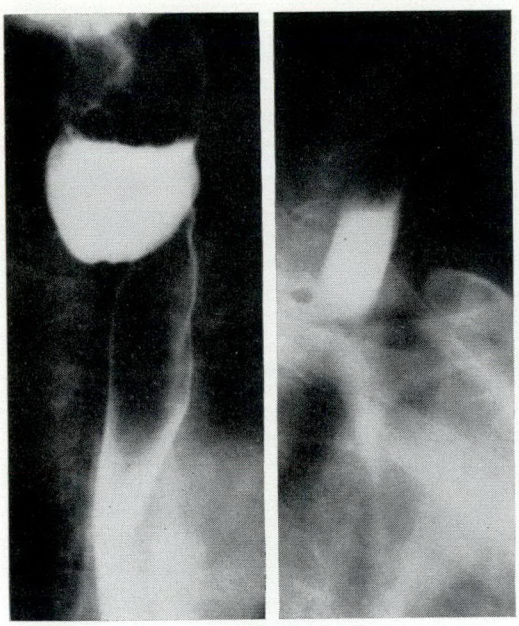

Fig. 2

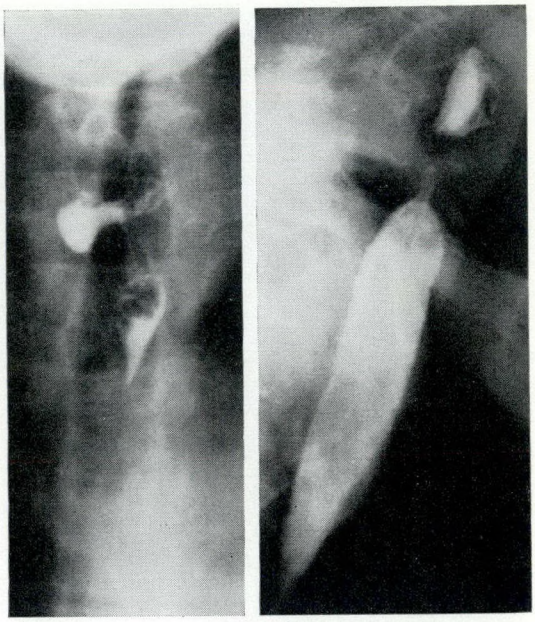

Fig. 3

Fig. 2.—Case 1.—Preoperative radiograph showing barium-filled pouch compressing and displacing the esophagus to the left and containing an air-fluid level. The lateral view shows a large residue of barium in the pouch after the passage of the bolus. **Fig. 3.**—Case 1.—Postoperative radiograph in the lateral view (right) shows no apparent remnant of the pouch with a normal-appearing upper esophagus. The anteroposterior projection shows a very small residue in the remaining fundus of the pouch retained by a small undivided remnant of the cricopharyngeus. The main portion of the pouch has been marsupialized into the lumen of the esophagus.

subsides quickly without treatment and is thought to be due to the burning rather than to any degree of infection. On the second postoperative day, sterile fluids were permitted orally, and on the fourth day a full soft diet was given. The patients swallowed without difficulty and developed no late complications. Antibiotics were not used systemically in either case.

In effect, this procedure marsupializes the diverticulum into the lumen of the esophagus, and the resulting scar formation stabilizes the pharyngeal ring somewhat so that elevation of the larynx again opens the esophageal lumen in an approximation of normal. The obvious advantages of the endoscopic method are its lack of postoperative complications in all cases reported to date, and the brevity of the procedure which allows its use in patients who could not survive a major procedure. Recurrence or the necessity for re-operation occurs in about 7% of cases as opposed to about 3% to 4% recurrence with surgical excision of diverticulum. However, the procedure may be repeated endoscopically as many times as necessary to achieve a good result. All patients have reported total or near-total relief of symptoms.

Case Reports

Case 1.—A.H., a 68-year-old man, was in good general health but gave a 12-year history of dysphagia and choking on solid food if it was eaten quickly and not well chewed. He frequently regurgitated food during meals, and after eating could evacuate the contents of the esophageal pouch by digital pressure on his neck. He had no associated pulmonary complications.

Barium swallow demonstrated the presence of a large Zenker's diverticulum (Fig. 2), and in February 1962 the Dohlman procedure was carried out.

On the first day after operation, intravenous therapy was discontinued, and sterile water per os was permitted. On the second day he was allowed full fluids, and on the fourth day was fed a soft diet. On the seventh day, he was eating a full diet without difficulty and was discharged from hospital. Two

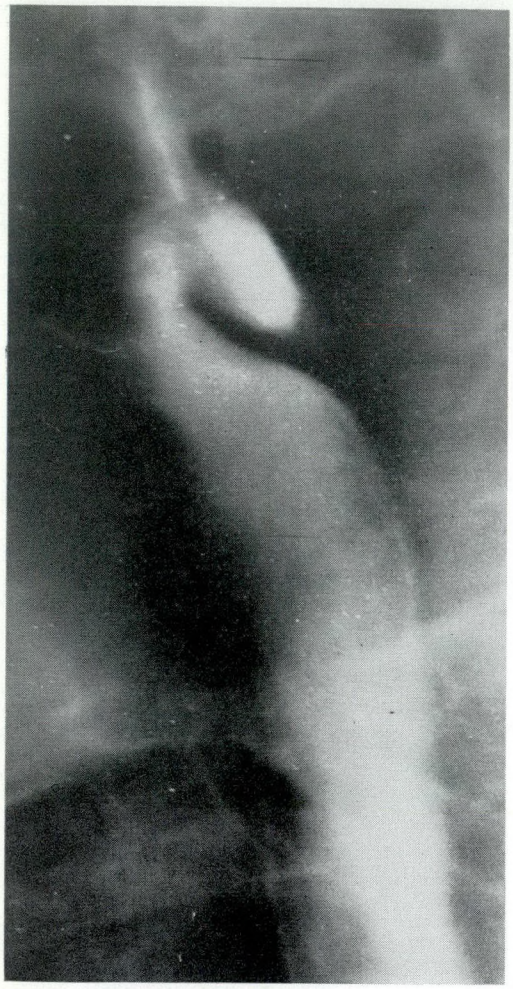

Fig. 4.—Case 2.—Preoperative radiograph showing characteristic appearance of a barium-filled diverticulum, measuring 2.0 cm. in length, compressing and displacing the lumen of the esophagus.

months later he was readmitted because although his swallowing was greatly improved and no regurgitation occurred, he still occasionally had the sensation of meat sticking in his throat. The Dohlman technique was again employed, and a further ¾" of the cricopharyngeus was divided (Fig. 3). On this occasion, he was advanced to a soft diet in three days. Subsequently he has experienced complete relief of symptoms.

On neither occasion were antibiotics used, and the patient did not develop the usual transient low-grade febrile reaction.

The operating time for each procedure was approximately 30 minutes.

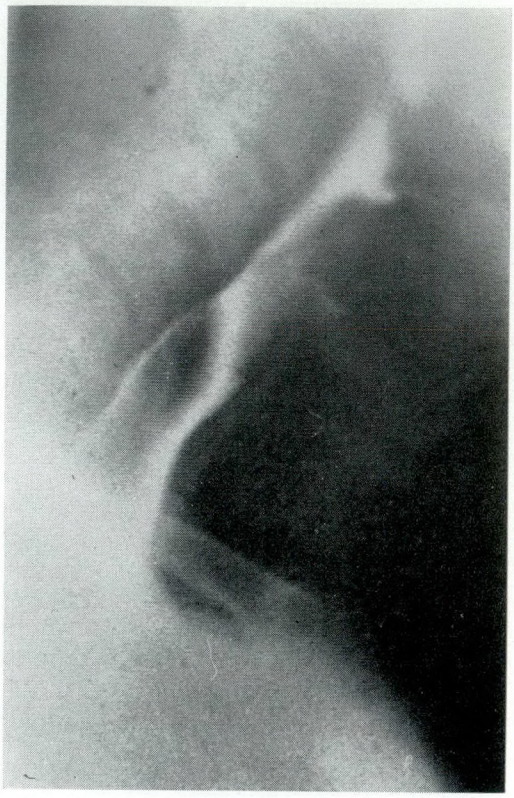

Fig. 5.—Case 2.—Postoperative radiograph showing no remnant of the diverticulum. This is a film taken during cinéradiographic examination. During this examination it was clearly shown that the bolus passed quickly through the cervical esophagus, and in this region the lumen was unusually large, demonstrating the incorporation of the diameter of the diverticulum into the total diameter of the postoperative esophagus.

Case 2.—C.P., a 73-year-old man, gave a two-year history of dysphagia with solid foods sticking in his throat. He also complained of regurgitation of undigested food anytime from one to two hours after eating. There had been no weight loss or pulmonary complications. His general health was good for his age.

Barium swallow demonstrated a moderate-sized Zenker's diverticulum (Fig. 4). An esophageal hiatus hernia was also demonstrated.

In December 1961 the Dohlman procedure was carried out. On the third postoperative day he was eating a soft diet without difficulty, and was released from hospital on the fifth day. For 24 hours postoperatively, the patient had a fever of 100.5° F. which promptly returned to normal spontaneously.

At a follow-up appointment five months later, the patient stated that he had no further

difficulty in swallowing, and no regurgitation. He still complained of belching, but this was attributed to the hiatus hernia.

Summary

These two cases are added to those previously reported from other centres to further demonstrate the simplicity of the Dohlman procedure, its effectiveness and its freedom from complications. Dohlman has recommended the procedure for patients who for various reasons related to age and general condition are not good surgical candidates. It is suggested that the technique might find application in a much broader selection of cases.

The authors wish to sincerely thank Professor G. Dohlman for his advice and instruction in this operation during his recent visit to Toronto as visiting Medical Research Council Professor to their department. They also thank Dr. G. E. D. Snell for his co-operation in the preparation of this case report, and Dr. I. B. Macdonald for allowing them to report his case.

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Fig. 6.—Case 2.—Postoperative film demonstrating the bolus just past the region of the diverticulum, without evidence of barium residue; strong evidence of complete obliteration of the sac.

Résumé

Les auteurs présentent ici deux cas de diverticule du pharynx traités par voie endoscopique selon la technique décrite par Dohlman. Ils démontrent ainsi la simplicité de cette technique, son efficacité et l'absence de complications qui la caractérise. Dohlman restreignait l'usage de sa méthode aux cas, qui pour des raisons diverses (âge, état général etc.), ne relevaient pas des thérapeutiques chirurgicales ordinaires. Les auteurs estiment que les indications de cette technique devraient être élargies.

CYSTIC DEGENERATION IN PERSISTENT CERVICAL THYMUS: A REPORT OF FOUR CASES IN CHILDREN*

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In a review of 124 cases of branchial cysts and sinuses treated at The Hospital for Sick Children between 1935 and 1960, four thymic "tumours" were found which presented clinical and morphological features differing from the more commonly occurring branchial cyst. The four cases occurred in males between the ages of seven and 13; the lesions were slow growing and lay along the carotid sheath without connection to adjacent structures (Fig. 1). They consisted of incompletely epithelialized cholesterol-containing cysts within solid thymic tissue. Although this cystic lesion was found in 3% of the lateral neck cysts and sinuses in this review, no such structures were described in over 600 cases of neck cysts and sinuses reported in the papers of Ladd and Gross,[1] Neel and Pemberton,[2] Rankow and Hanford[3] and Lyall and Stahl.[4]

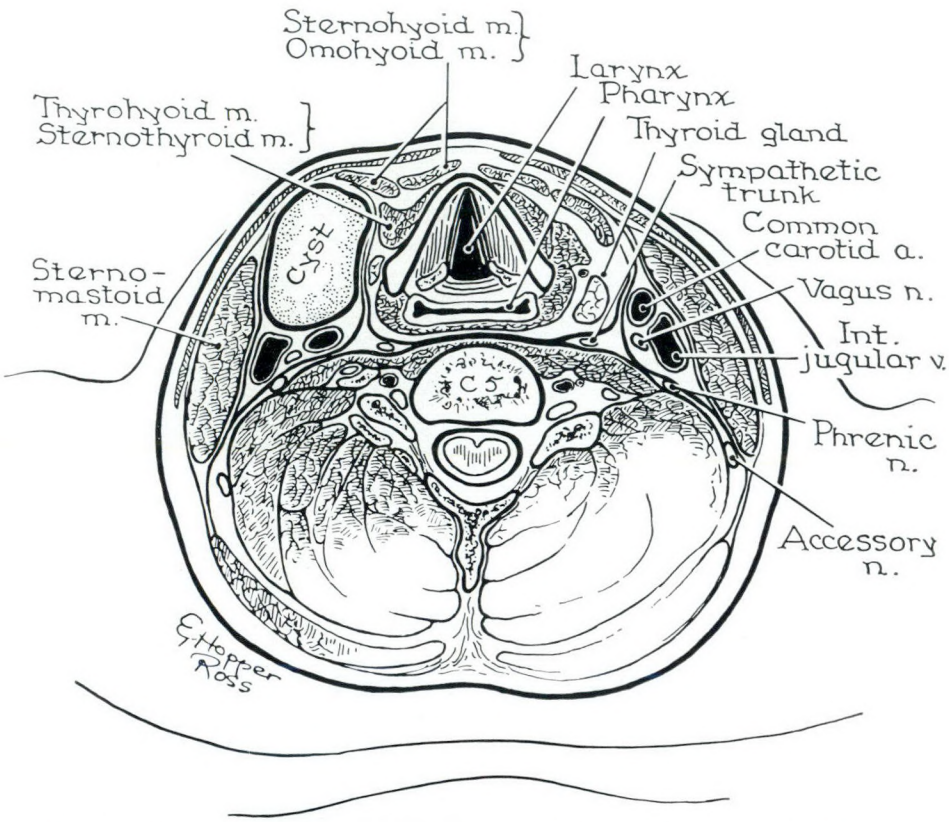

Fig. 1.—Diagrammatic cross-section of the neck to show the position of the lesion.

Small nodules of thymic tissue and cysts are not infrequently seen in the neck at autopsy. In a review of the literature, however, only 10 cases were found where such

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TABLE I.—Summary of Reported Cases of Masses of Cervical Thymic Tissue Containing Cysts, Including the Four Cases in this Report

Author	Age (years)	Sex	Side	Duration	Size (cm.)	Location
1 Pollosson and Piéry[5] (1901). Quote from Côté and Fortin..	1½	M	R	—	—	———
2 Gilmour[6] (1939). Case of R. Wright	18	F	—	3 mo.	10 cm.	Bifurcation of carotid artery
3 Hyde *et al.*[7] (1944)	5	M	R	8 mo.	7 x 5 x 3.2	Bifurcation of carotid artery
4 Weller *et al.*[8] (1951)	4½	F	L	4 mo.	7 x 4.5 x 4	Along carotid sheath
5 Williams and Gerber[9] (1957)..	6	M	L	Several weeks	12 x 5 x 5	Along carotid sheath
6 Crawford *et al.*[10] (1957)	6	M	R	3 yr.	3.5 x 3 x 2	Along carotid sheath
7 Willis[11] (1958)	31	M	—	—	—	———
8 Viar[12] (1959)	6	M	L	4 yr.	15 x 6	Along carotid sheath
9 Lane[13] (1960)	27	F	—	Several weeks	2.0 cm. diameter	Along carotid sheath
10 Côté and Fortin[14] (1961)	6	F	R	6 days	—	Along carotid sheath
11, 12, 13, 14 Present report: Fielding *et al.*	7	M	L	4 yr.	7 x 4 x 3.5	Along carotid sheath
	7	M	R	3 mo.	17 x 4 x 2	Along carotid sheath
	13	M	R	4 mo.	11 x 4 x 2.5	Along carotid sheath
	8	M	L	3 days	15 x 5 x 2	Along carotid sheath

a lesion attained a clinically recognizable size.[5-14] These are listed in Table I and so closely resembled the four cases in our series that these lesions appear to constitute a distinct entity. Most of the cases have been reported in recent years, and this lesion is probably more common than the literature would indicate.

Case Reports

Case 1.—At the age of three, this seven-year-old boy fell from his tricycle striking his neck on a cement block. A swelling, described as being the size of a grapefruit, formed in the left side of the neck, but over the next year decreased to the size of an egg. At operation, it was found to lie deep to the platysma

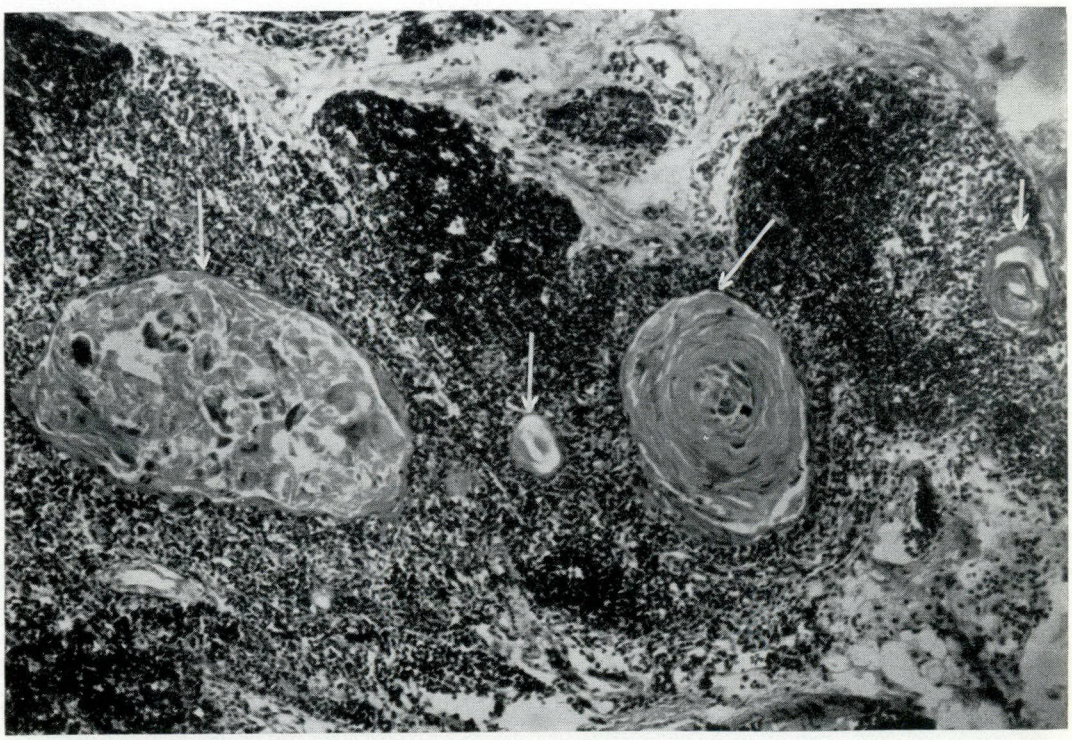

Fig. 2.—Case 1. Photomicrograph showing degenerative changes and enlargement of Hassall's corpuscles (see arrows). x 100.

Fig. 3.—Case 1. Photomicrograph showing a large cystic space containing cholesterol crystals, an epithelial lining, an area of thymic tissue containing Hassall's corpuscles and an area of granulation tissue (right). x 40.

and deep fascia and was shelled out without difficulty. The surgical specimen was a soft lobulated mass measuring 7 x 4 x 3.5 cm. The cysts, which measured up to 2 cm. in diameter, contained a cloudy fluid and the thin walls were partly lined by flattened epithelium (Figs. 2 and 3). Many areas showed involuted thymic tissue.

CASE 2.—This seven-year-old boy had noticed a lump in the right side of the neck three months before admission to hospital. It was soft, non-tender and lay deep to the mid-portion of the sternomastoid; it did not move on swallowing.

At operation it was found to lie beneath the deep fascia, sternomastoid and strap muscles. The upper part extended upwards to the hyoid bone and formed a bluish-green cyst. Removal was from above downward, separating the mass from the carotid sheath posteriorly, the thyroid cartilage and trachea medially, and the sternomastoid laterally. The thyroid gland was not exposed. The mass con-

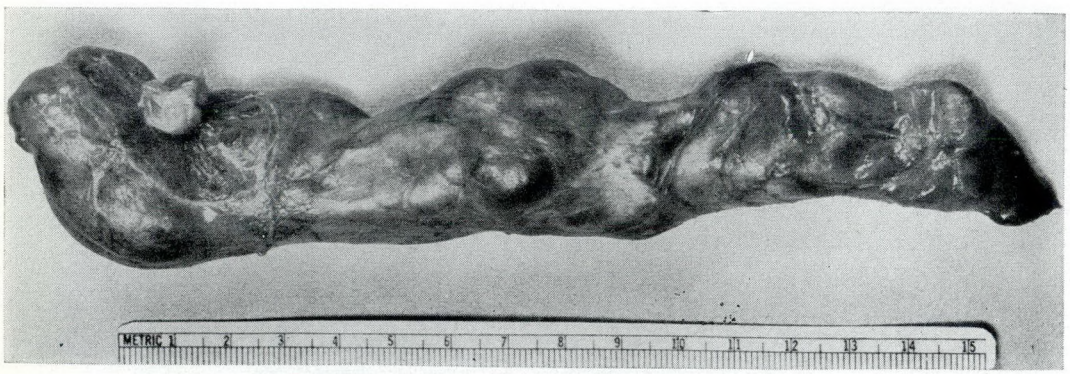

Fig. 4.—Case 2. Gross specimen. Note complete capsule.

Fig. 5.—Case 2. Photomicrograph showing thymic tissue, areas with cholesterol clefts and a foreign-body giant-cell reaction, a small cyst and infolding of the lining of the large cyst. x 40.

tinued downward to the level of the manubrium sterni where it was easily separated from the thymus.

The upper 13 cm. of the gross specimen (Fig. 4) showed multilocular cysts containing a greenish-brown fluid with cholesterol crystals. The lower portion was not cystic, but was composed of solid thymic tissue. The cysts were incompletely lined by flattened epithelium and the walls contained vascular fibrous tissue with cholesterol clefts and many foreign-body giant cells (Fig. 5).

Case 3.—This 13-year-old boy had noted a 4 cm. painless swelling on the right side of the neck following a cold, four months before admission to hospital. The mass was soft and cystic, and moved on swallowing or coughing. The trachea was displaced to the left.

As it was felt preoperatively that this was a thyroid adenoma, the approach was made through a Kocher incision. The mass lay beneath the deep cervical fascia, sternomastoid and strap muscles and was lateral to and separate from the thyroid gland. Posteriorly, it was loosely adherent to the carotid sheath from the bifurcation of the carotid artery downward almost to the subclavian artery.

The mass was well encapsulated and measured 11 x 4 x 2.5 cm. (Fig. 6). The cysts were smooth-walled and contained a watery fluid with shimmering cholesterol crystals and amorphous debris. On microscopical examination, thymic tissue and a few fragments of parathyroid were identified lying between cystic spaces, the smallest of which were slightly larger than a Hassall's corpuscle. The lining of the cysts was predominantly granulation tissue with a few small areas of squamous and transitional epithelium.

Case 4.—Three days before admission to hospital, an asymptomatic mass was found in the left side of the neck of this otherwise healthy eight-year-old son of a physician. It was soft and lay at the anterior border of the sternomastoid opposite the upper edge of the thyroid cartilage. The mass did not move on swallowing. The trachea was displaced to the right (Fig. 7).

The mass was found to lie deep to the cervical fascia, strap muscles and anterior edge of the sternomastoid but superficial to the carotid sheath. It was nodular, bluish-grey in colour and encapsulated (Fig. 8). The upper end was 5 cm. in diameter and ex-

Fig. 6.—Case 3. Gross specimen which has been opened to show the multilocular cysts.

tended to the hyoid bone, where it ended without evidence of an ascending tract. Below, at the level of the thyroid cartilage, it narrowed to 2 cm. in diameter and continued down the neck to the upper edge of the manubrium where it again ended blindly and without evidence of a connection with the normal thymus gland. The mass was closely adherent through its entire length to the carotid sheath. It lay lateral to the thyroid gland, adherent to it, but separated by a distinct fascial plane. The upper portion had separated the carotid artery and the jugular vein.

The cut section of the specimen showed numerous irregular cystic spaces filled with a brownish shimmering fluid. The cyst walls varied from 0.3 to 0.5 cm. in thickness. Microscopical examination showed thymic tissue with many cholesterol-containing cysts sur-

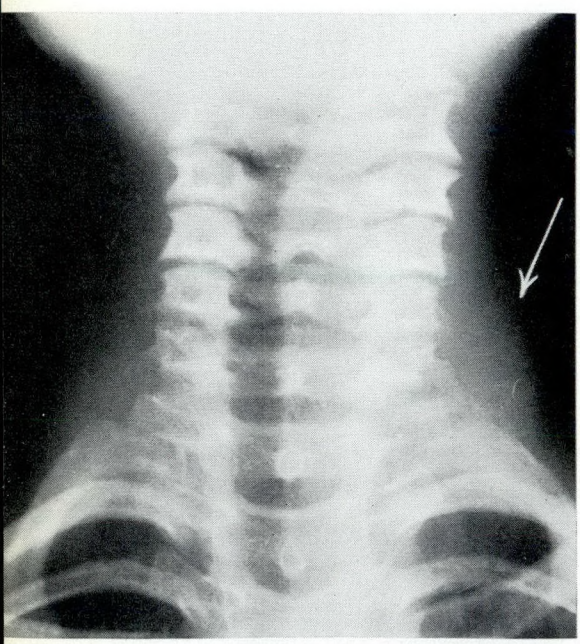

Fig. 7.—Case 4. Radiograph of the neck showing the soft tissue mass on the left with displacement of the trachea to the right.

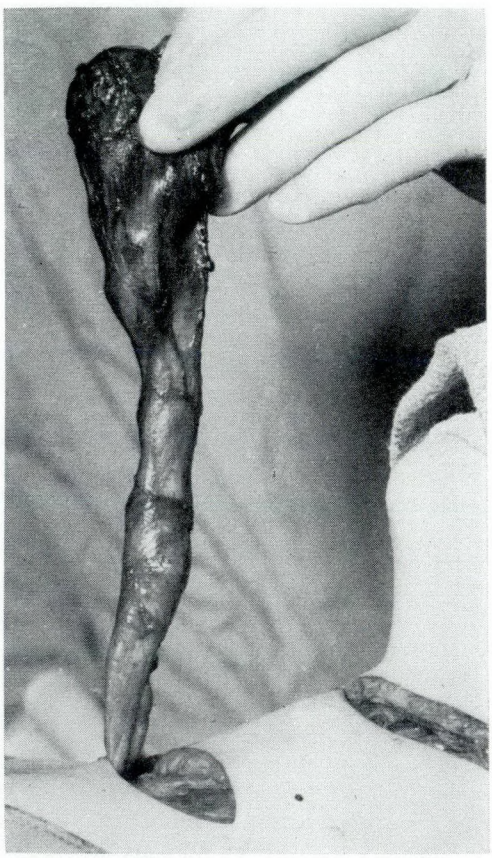

Fig. 8.—Case 4. Showing the mass being removed from the neck through "step-ladder" incisions.

Fig. 9.—Case 4. Photomicrograph showing (centre) normal thymic tissue, cholesterol clefts and foreign body reaction (left) and liquefaction and cyst formation (right). x 16.

rounded by fibrous tissue and a giant-cell reaction (Fig. 9).

Summary of Cases

The four cases showed a remarkable similarity of clinical, anatomical and pathological features. The patients were between the ages of seven and 13 years, the lesions were of relatively short duration and were essentially asymptomatic. Surgical removal was not difficult; there were no complications, and there have been no recurrences in two to 10 years follow-up. All of the lesions occupied a similar position in the neck (Fig. 1), and showed no apparent connections with other structures or with the normally located thymus gland. They were cylindrical in shape, 7.0 cm. to 17.0 cm. in length and encapsulated, and contained many multiloculated cysts filled with fluid and cholesterol crystals. The cysts were lined with granulation tissue and squamous epithelium and were surrounded by a chronic inflammatory reaction. Thymic tissue was present within the mass in all four cases. A normal thymus gland was stated to be present in two of the cases and not commented upon in the others.

Discussion

The thymus gland is generally believed to arise in the sixth week as an outpouching of the third branchial pouch.[15, 16] This outpouching is at first hollow, but forms a solid strand as it migrates down the neck. It passes medially to lie just behind the developing thyroid gland. It becomes adherent to the pericardium and descends with the pericardium into the chest. Norris[17] states that it picks up an epithelial component from the nearby cervical sinus. The tract usually disappears by the third month but may persist in part. The primordium of the thymus gland forms a bilobed mass of closely packed epithelial cells which becomes infiltrated by mesodermal tissue and lymphocytes. Although the origin of Hassall's corpuscles is still in dispute, they are generally believed to be the fragmented squamoid foci of the epi-

thelial cells within invading mesoderm. The gland, therefore, has an ectodermal and a mesodermal component.

Abnormalities in the location of the thymus may be due to partial or complete failure of descent into the thorax. Partial failure leads to persistence of thymic tissue within the neck or a persistent cervical tail with a normally placed thymus.[18] Pirkey[19] described a tumour occurring in a persistent cervical thymus gland in an 11-year-old girl.

If the upper end of the pharyngolymphatic duct fails to regress in the eighth week, sequestrated cystic or solid nodules could be left along the course of migration. Small nodules of thymic tissue and columnar epithelial-lined cysts are found incidentally around the thyroid in some autopsies on infants.[20] Wenglowski,[21] in 1913, reported a series of autopsies in which he found small thymic cysts and rests in the necks of two out of 10 adults and 21 out of 65 infants. Gilmour[18] reported 13 cases and King[23] reported eight cases where small cysts with thymic tissue were found in the neck at autopsy.

Thymic tissue may, therefore, occur in the neck as separate nodules of mature well-differentiated thymic tissue or in association with ciliated or columnar epithelial-lined remnants of the pharyngeal outpouching.[24] Serial blocks of the surgical specimens were cut and examined for such ciliated or columnar epithelium but none was found. It was noted, however, that stages in the development of the cysts from Hassall's corpuscles could be seen in each specimen. In Figs. 2, 3, 5 and 9, for example, normal-appearing Hassall's corpuscles are seen adjacent to corpuscles showing central necrosis and cyst formation.

The close resemblance between the microscopical appearance of the cystic portions of these lesions and the centre of Hassall's corpuscles suggested that they arise from the Hassall's corpuscles within aberrant nodules of thymus gland in the neck and not directly from the epithelium of the pharyngeal outpouching. The great variety of pathological features seen in the masses is probably secondary to degenerative changes in the epithelial component of Hassall's corpuscles, which is derived either from the thymic pouch or the cervical sinus. If these degenerative changes are extensive enough, a cystic dilation of the channels forming the Hassall's corpuscles would occur, producing a gradual destruction of the epithelium lining these cysts. Ruptures in the wall surrounding these cysts with seepage of the contents into the tissues would cause a foreign body reaction, and would explain the large numbers of giant cells with some plasma cells, lymphocytes and fibrosis which are commonly seen in these lesions. These processes have been described by Willis[11] and Castleman.[25]

No definite ciliated or columnar epithelium was found in this series, although it is commonly seen in the small vesicular canalicular and gland-like structures in the neck as described by Willis,[24] Gilmour[20] and others. These structures probably represent persistent pharyngothymic ducts, intrathymic dermoids, thyroglossal ducts or parathyroid anlagen and are congenital and distinct from the apparently acquired thymoepithelial cysts described in this report. Willis[26] suggests that the ciliated epithelium in the smaller type of cysts may arise either by metaplasia of already differentiated epithelium of Hassall's corpuscles or as a primary developmental heteroplasia.

With the exception of trauma occurring in one patient and an upper respiratory infection in another, no etiology for these tumours was apparent. The age incidence coincided with the age of maximal rate of involution of the thymus gland. There was no suggestion of myasthenia gravis in any of the cases, a condition occasionally associated with thymic cysts. The preponderance of males in this series is also at variance with the preponderance of females in myasthenia gravis. It would appear that aberrant thymic tissue in the neck has a greater tendency to form cysts than has the normal gland.

The cysts in this report appear to be unrelated to the more common branchial cleft cysts and sinuses arising from the upper branchial arch structures. Wenglowski[21] and others have maintained that the typical branchial cyst or sinus arises from the third

pouch and cervical sinus, but it is more generally felt that they are of the second cleft and pouch origin. Although the common branchial cysts and sinuses are usually surrounded by lymphoid tissue, they are lined with ciliated and columnar epithelium and characteristically lie along a line extending from the skin in the lower neck, through the carotid bifurcation to the upper tonsillar fossa. However, the thymo-epithelial cysts described in this paper are deep to the clavicle and remain lateral to the carotid sheath.

McGoon[27] has discussed the literature on midline teratomas which are more complex developmental anomalies than those reported here and although they may contain thymic tissue should be considered as a separate condition. Similarly, cystic masses in the neck as reviewed by Tenery, Abul-Haj and Burt,[28] appear to be different from our cases, as do neck choristoma.[22]

Summary

Four cases of an unusual lateral cystic lesion in the neck treated by excision are described. The lesions were asymptomatic, lay along the carotid sheath, and occurred in boys between seven and 13 years of age. The surgical specimens measured up to 17 x 5 x 3.5 cm. It is suggested that these lesions develop as degenerative changes in the Hassall's corpuscles of aberrant cervical thymic tissue and constitute a clinical entity.

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Résumé

Dans une étude statistique portant sur 124 cas de kystes branchiaux traités au "The Hospital for Sick Children" de Toronto entre 1935 et 1960, on trouve quatre "tumeurs thymiques" qui présentaient des caractères cliniques et morphologiques bien spéciaux. Ces quatre cas concernent des garçons entre sept et 13 ans. Les lésions consistaient en kystes épithélioïdes remplis de cholestérol situés dans le parenchyme thymique. Ces tumeurs furent relativement asymptomatiques; l'ablation chirurgicale en fut facile et l'on n'observa pas de récidive dans le période post-opératoire de 10 ans. L'étiologie de cette affection relève probablement d'un trouble dans l'embryologie; les anomalies de position du thymus sont dues à une descente incomplète de l'organe dans la cage thoracique. Dans certains cas, une persistence de petits ilôts de tissu thymique peut exister dans la région du cou. Ces ilôts se présentent, du point de vue histologique, soit comme des petites masses de tissu thymique normal, soit sous forme kystique. Dans la dernière éventualité, le tissu thymique est généralement associé avec des résidus épithéliaux cylindriques ciliés. Du fait de l'apparence microscopique très particulière que peuvent prendre ces formations, les auteurs pensent qu'il n'est pas exclu qu'elles trouvent leur origine à partir des corpuscles de Hassall.

SURGERY OF THE CHEST. Edited by John H. Gibbon, Jr. 902 pp. Illust. W. B. Saunders Company, Philadelphia; McAinsh & Co. Ltd., Toronto and Vancouver, 1962. $29.20.

It is becoming increasingly difficult to produce a textbook of thoracic surgery at a time when many aspects of this field are developing or changing very rapidly. In his Surgery of the Chest, John Gibbon has edited a multiple-author text which provides an excellent review of current knowledge and concepts of management of thoracic surgical problems. The subject material is comprehensive and appears in a variety of literary styles; in each instance the responsible author is well known because of his interest and experience in his particular subject.

Recent advances in cardiopulmonary physiology are clearly and concisely presented. In particular the physiology and surgical problems relating to total extracorporeal circulation and hypothermia with extracorporeal circulation are very well covered.

The clinical aspects of chest surgery generally are well and fairly dealt with; the sections dealing with suppurative and fungal diseases of the lung, the pleura and artificial heart valves are particularly excellent and complete. Separate chapters on endoscopy and radiological diagnosis provide useful information which is not infrequently neglected in such a text.

As in all multiple-author texts, there is considerable variability in the objectivity and value of any given section. In some chapters, undue priority is given to American contributors. This is a minor defect, however, and a number of the sections include a very comprehensive and contemporary review of the literature, which may be useful for reference work.

The book is an excellent one, and is highly recommended to anyone with an interest in the surgery of the chest.

SMOOTH MUSCLE TUMORS OF THE ALIMENTARY TRACT. John E. Skandalakis and Stephen W. Gray with Duncan Shepard and Geoffrey H. Bourne. 468 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1962. $19.25.

As the authors point out in their introduction, tumours of smooth muscle are so infrequent in any single surgical practice that few persons become experts in the diagnosis. This very fact justifies the publication of the present monograph in which a large number of reported cases are gathered from the literature and thoroughly reviewed. The only alternative might have been to leave the whole situation alone in the manner of an ostrich burying its head in the sand. The authors are to be congratulated for their singleness of purpose in compiling this book in spite of the easy alternative.

Of its very nature, this book cannot be particularly exciting; yet, for those who discover smooth muscle tumours in the alimentary tract (from the esophagus to the large intestine) even infrequently, it will be most valuable. Criteria for malignancy and the problem of borderline cases are described and basic guidelines to correct preoperative diagnoses are established. The views of the authors arising from their study should gain wider currency. They recommend treating leiomyosarcomas as true cancers requiring wide resection and a meticulous search for metastases.

More than 200 pages are devoted to an analysis of individual case reports from all the available literature on the subject. Tedious as these are to the casual reader, they provide a matchless body of information. The bibliography runs to 850 individual reports and attests to the thoroughness of the two senior authors. These features alone make this new book a valuable addition to the literature on cancer.

CASE REPORTS

TUMEUR GLOMIQUE DE L'ESTOMAC*

EUCHARISTE SAMSON, M.D., F.A.C.S., WILLIAM BASTARACHE, M.D. et JEAN-LOUIS BONENFANT, M.D., F.R.C.P.[C], *Québec, P.Q.*

Le glomus ou corps glomique est une anastomose artério-veineuse, d'un type particulier, que l'on rencontre surtout au niveau du tissu conjonctif sous-cutané des extrémités. Il sert à régler le débit des artères terminales, et, de ce fait, l'homéostasie thermique des extrémités, et peut-être agirait sur la tension artérielle. Il se compose d'une artère afférente qui devient brusquement le segment artériel, à lumière étroite, à paroi épaisse formée de cellules épithélioïdes et entourée d'un manchon de fibres nerveuses sensitives et vaso-motrices. Ce segment peut, au besoin, se dilater par l'amincissement et l'allongement des cellules pariétales. Plus loin, la lumière s'élargit soudain et n'est plus entourée que d'une gaine fibreuse: c'est le segment veineux qui débouche dans la veine afférente. Le corps glomique se compose souvent de plusieurs canaux plus ou moins contournés. Le tout baigne dans une masse de tissu conjonctif lâche, bien délimité, qui tranche sur le derme fibreux.[4]

La tumeur glomique est décrite sous plusieurs termes: tubercule cutané douloureux, périthéliome, endothéliome, angioneuromyome artériel, glomus neuro-vasculaire de Masson[6] ou encore glomangiome de O'Bailey.[7] C'est une hyperplasie nodulaire d'une des structures que nous venons de décrire. Peu fréquente, on la rencontre surtout dans la région sous-unguéale, palmaire ou plantaire, rarement sur les avant-bras ou les jambes, et encore plus rarement sur le tronc. Macroscopiquement, cette tumeur est de taille variable, bien isolée et souvent encapsulée. Elle est de consistance molle, de coloration rosée ou parfois jaunâtre; à la coupe, elle est très hémorragique.

L'aspect microscopique est caractéristique. Des cellules normales tapissent des espaces vasculaires de dimensions variées. Ici et là, dans ces espaces, on remarque des amas de tailles inégales composés de cellules épithélioïdes rondes. Ces cellules ont un petit noyau central foncé et un cytoplasme peu abondant, clair, presque limpide. Bénigne, cette tumeur se traite par l'ablation chirurgicale simple, et son pronostic est excellent.

Observation

R.A., un homme âgé de 69 ans, fut admis à l'Hôtel-Dieu de Québec, le 26 mai 1961. A son arrivée, il se plaignait de douleurs épigastriques prenant la forme de brûlures ayant débuté un an auparavant, que ni les aliments, ni les alcalins ne pouvaient soulager. Cette douleur s'irradiait vers le dos et présentait, sur un seuil constant, des périodes d'exacerbation qui empêchaient le patient de dormir.

Depuis un mois, le patient avait des vomissements après chaque repas; il prétextait que les aliments solides ne pouvaient passer, et devait se contenter de liquides. Il n'a jamais présenté ni melaena, ni hématémèse et conserva son appétit. Son histoire médicale antérieure était sans rapport avec la présente affaire.

L'examen physique montra un patient amaigri mais, par ailleurs, peu souffrant. Sa tension artérielle était à 130/70 mm. Hg et son pouls à 80/minute. L'examen cardio-vasculaire ainsi que l'examen abdominal s'avérèrent négatifs. Il ne présentait aucune lymphadénopathie. Au toucher rectal on nota cependant une hypertrophie prostatique.

Examens de laboratoire.—Les résultats des examens de laboratoire furent les suivants: hémoglobine, 14.7% mg.; hématocrite, 44; globules blancs, 8,750 mm.^{3}; compte différentiel, normal; azote uréique, 0.170 g. %; urée, 0.364 g. %; examen d'urine, normal sauf pus + et *Trichomonas vaginalis.*

Les radiographies de l'estomac montrèrent un ulcère sténosant de la petite courbure, dans la région pré-pylorique sans aucun indice d'autre pathologie (Fig. 1).

Une intervention chirurgicale fut pratiquée. Au cours de l'exploration, on palpe, à la partie distale de la grande courbure de l'estomac,

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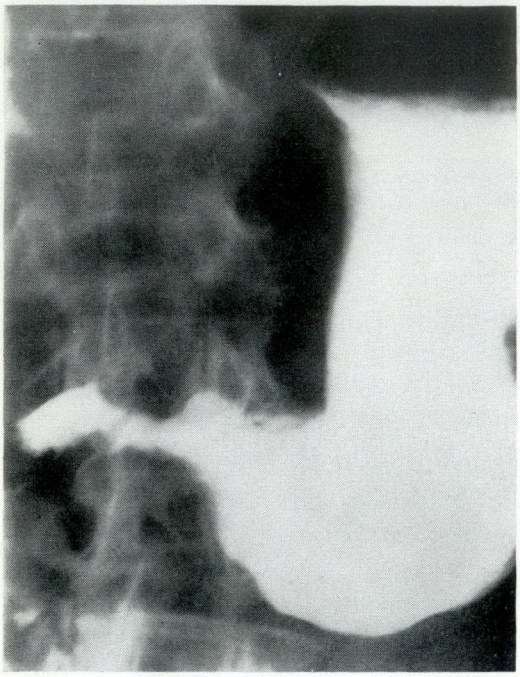

Fig. 1.—Présence d'une niche sur la petite courbure pré-pylorique.

un nodule tumoral assez bien délimité, intra-pariétal, de 3 x 2.5 x 2 cm. On résèque ce nodule et à l'examen extemporané, le pathologiste décrit une tumeur richement vascularisée pouvant correspondre à une tumeur glomique. Une gastrectomie sub-totale est ensuite effectuée pour une ulcération de 1.5 cm., située sur la petite courbure, à la région pré-pylorique (Fig. 2).

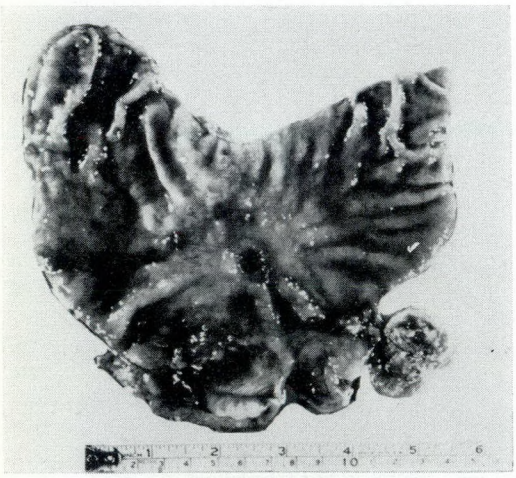

Fig. 2.—Estomac ouvert le long de la grande courbure, montrant l'ulcère de la petite courbure. A la partie droite de la pièce opératoire, le nodule correspond à la tumeur intra-pariétale.

La tumeur intra-pariétale a une forme ovoïde, une consistance molle; la surface de coupe est jaunâtre avec de multiples foyers hémorragiques.

Examen histologique.—L'ulcération est tapissée par un épais liséré fibrino-nécrotique et purulent. La paroi gastrique sous-jacente est constituée par du tissu de granulation riche en filets nerveux hyperplasiques, infiltré de nombreux polynucléaires éosinophiles. Dans la muqueuse du voisinage on trouve un infiltrat inflammatoire chronique marqué et plusieurs glandes sont de type intestinal. Dans la proximité immédiate de l'ulcère on observe plusieurs cellules épithéliales atypiques avec des noyaux irréguliers, monstrueux, parfois en mitose; ces modifications cellulaires correspondent vraisemblablement à une transformation épithéliomateuse *in situ*.

La tumeur prélevée dans la paroi gastrique correspond à un néoplasme développé dans la sous-muqueuse, généralement bien délimité, bien que dépourvu de capsule propre; cette tumeur est constituée de très nombreux vaisseaux sanguins, de taille très variable, dont la paroi est de nature soit musculaire, soit purement endothéliale (Fig. 3). Tout à fait à proximité de ces structures vasculaires, il y a des plages et des traînées de cellules rondes, à chromatine dense, dont l'aspect correspond à celui de la cellule glomique. On trouve aussi de nombreux macrophages bourrés de pigments sanguins (Figs. 4 et 5). Le néoplasme a un stroma scléreux très abondant, par endroits très peu cellulaire.

Cette tumeur gastrique correspond à une tumeur glomique.

Discussion

Si la tumeur glomique des extrémités est relativement fréquente, celle de l'estomac est extrêmement rare.[1, 2, 7] En effet, on n'en trouve que 11 cas dans la littérature médicale jusqu'à présent. Les trois premiers furent publiés par Kay et ses associés[5] en 1951. Une étude comparée des neuf premiers cas, effectuée par Donovan, Graham et O'Donnell de Boston,[3] a démontré que cette tumeur se rencontre surtout entre quarante et soixante-dix ans, avec une prédominance de trois pour un en faveur du sexe masculin. De taille variable et souvent intra-murale, c'est une tumeur qui se rencontre habituellement à la région pré-pylorique et dont la symptomato-

Fig. 3.—Aspect angiomateux du néoplasme (x 100).

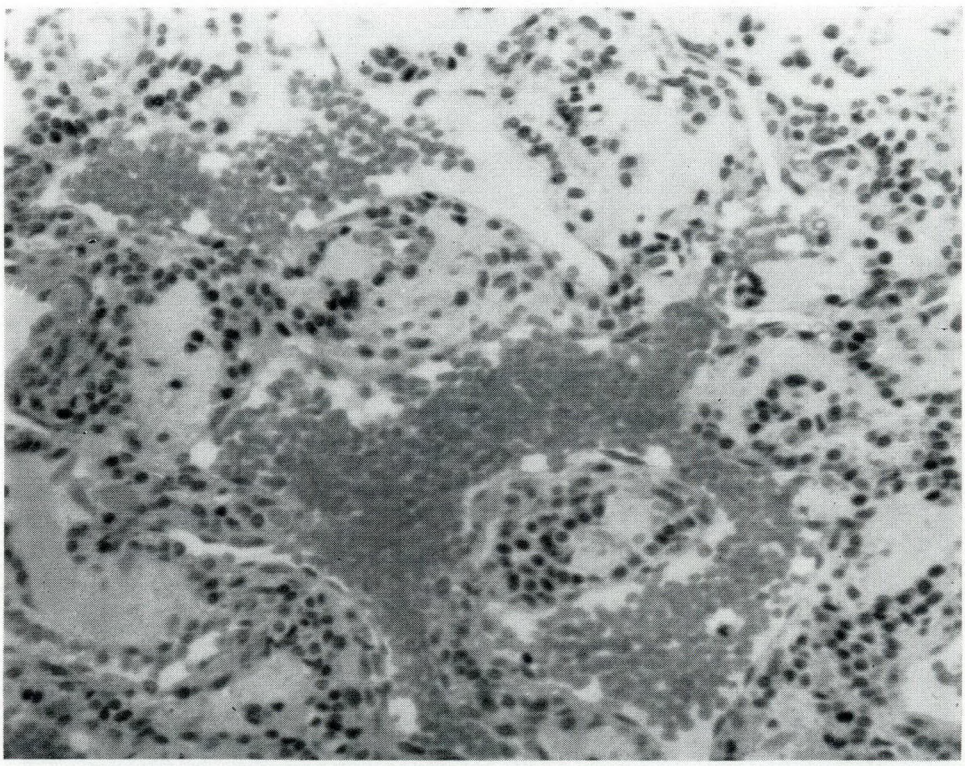

Fig. 4.—Présence de petites cellules à noyau très dense, du type de celui de la cellule glomique (x 200).

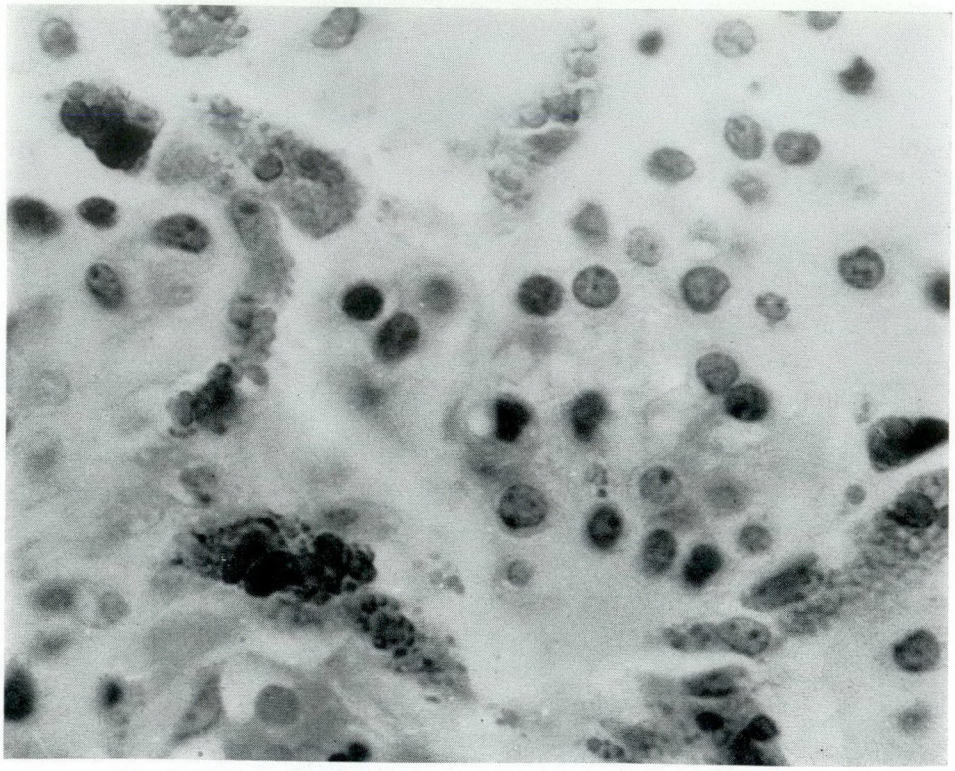

Fig. 5.—Présence de macrophages bourrés de pigments sanguins (x 400).

logie peu caractéristique peut se confondre aisément avec celle de l'ulcère gastrique qui l'accompagne parfois. Quatre patients sur neuf souffrirent d'hématémèse et de choc consécutifs à l'ulcération de la tumeur dans la lumière gastrique.[3] Dans cinq cas, on a pu identifier la tumeur à l'examen extemporané, tandis que dans trois cas, la radiologie a permis le diagnostic.

Dans le cas que nous présentons, la tumeur fut trouvée par hasard, dans la paroi gastrique, au niveau de la partie distale de la grande courbure, au cours d'une gastrectomie pour ulcère peptique sténosant.

Conclusion

Un cas de tumeur glomique de l'estomac découvert au cours d'une laparotomie pour ulcère gastrique est rapporté. Relativement fréquente aux extrémités, cette tumeur est cependant considérée comme très rare au niveau de l'estomac. Les caractères histologiques sont suffisamment typiques pour éviter la confusion avec un autre néoplasme.

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Summary

A case of a glomus tumour, localized in the gastric wall and discovered during an exploration for gastric ulcer, is reported. This type of neoplasia is relatively frequent in the subcutaneous connective tissue of the extremities of the limbs; it is, however, seldom found in the stomach. The histological features are very characteristic and allow a precise diagnosis so that no confusion with any other type of tumour is possible.

CAROTID CAVERNOUS FISTULA WITH PULSATING EXOPHTHALMUS: A Fortuitous Cure

DWIGHT PARKINSON, M.Sc., F.R.C.S.[C], F.A.C.S.* and ROBERT M. RAMSAY, M.D., M.Sc.,† *Winnipeg, Man.*

THE dramatic clinical entity known as carotid cavernous fistula with pulsating exophthalmus has been a challenge and a puzzle from the beginnings of neurosurgery. It has been known for many years that some undergo spontaneous recovery. Some patients are cured by simple carotid ligation in the neck,[1, 2, 4, 9] although others may be killed by the same procedure.[6] Echols and Jackson[5] and Jaeger[8] present excellent reviews of the various methods by which cure of this condition has been attempted over the years.

The following case is unusual in that the cure was quite fortuitous.

CASE REPORT

A 12-year-old boy fell from a tractor and struck his left orbit on his left knee. He was not rendered unconscious but noted immediately that he was blind in the left eye. He was examined very shortly thereafter by the authors. The left eye was blind with no direct pupillary response but normal consensual response. The fundus and the optic nerve head were normal. There was no discolouration or swelling of the lids or conjunctiva. The remaining neurological examination was entirely normal. A bruit synchronous with the heart beat was easily audible to the examiner; the patient at first denied but later admitted hearing this bruit. Angiography revealed that the contrast media from the left internal carotid was shunted into the cavernous sinus. Following angiography the bruit disappeared and did not return during a week of observation. The boy was allowed to go home. The left eye remained blind and subsequently optic atrophy developed. It was assumed that he had sustained a fracture through the left optic foramen although this could not be demonstrated by radiographic examination.

Less than a month later the boy returned with a left pulsating exophthalmus and a bruit audible to both the patient and the examiner (Fig. 1). Primary optic atrophy was present on the left. Panangiography again revealed the presence of a fistula from the left carotid to the cavernous sinus, which in turn drained through a large tortuous pathway into the left orbit. There were numerous other venous channels along the base through which the contrast media dispersed also (Fig. 2a). The contents of the right carotid spilled across into the shunt without contralateral compression. With contralateral carotid compression, the right carotid irrigated both hemispheres as well as the shunt (Fig. 2b). The vertebral system could not be demonstrated to spill into the shunt but would fill the left carotid branches distal to the shunt (Fig. 2c). There was no abnormal connection between the vertebral system and the carotid system such as a persistent trigeminal artery.[7]

At operation the internal carotid artery was exposed in the neck and then clipped intracranially above the clinoid process. The ophthalmic artery was exposed by unroofing a portion of the orbit and retracting the optic nerve medially. The ophthalmic artery was ligated with ease after which the internal carotid was ligated in the neck. To our dismay the pulsations and bruit persisted. All three points of occlusion were re-checked and additional ligatures were placed at each point. There was no change in the pulsations or the bruit. It was evident that in this patient, over a period of less than one month, a vigorous collateral had developed to this trapped segment of the internal carotid through pre-existing channels. The internal carotid was then opened above the ligature in the neck. It was our intention to introduce a piece of muscle on a suture and float it up to the carotid siphon as an embolus, hoping it would lodge in the fistula.[8] However, the arterial backflow from this trapped segment was so forceful that there was considerable doubt as to the direction in which an embolus might float, even after the release of the proximal ligature on the internal carotid. It was then decided to introduce a catheter through the incision in the hope that it would pass beyond the carotid siphon and carry with it a ligature which could be grasped through an incision in the carotid above the clinoid. This ligature would be used to pull a strip of muscle up through the carotid into the region of the

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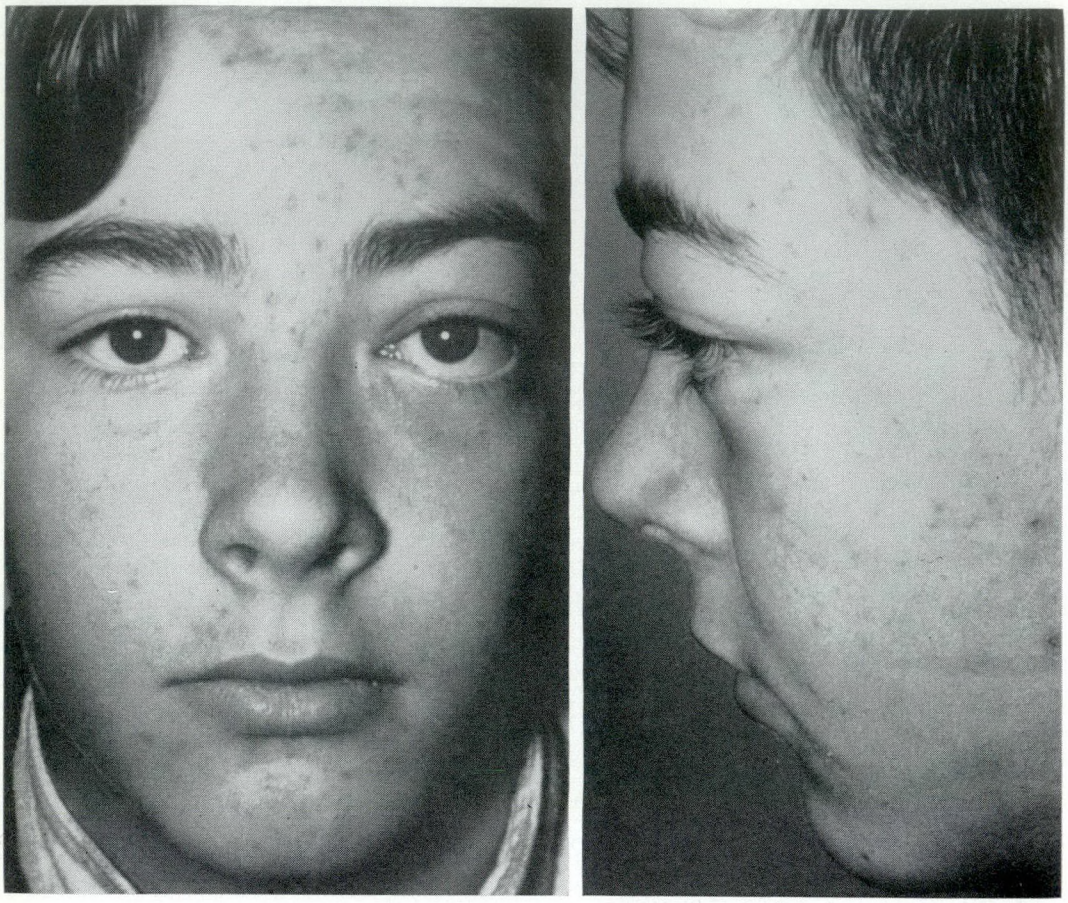

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Fig. 1.—Views of patient, preoperatively.

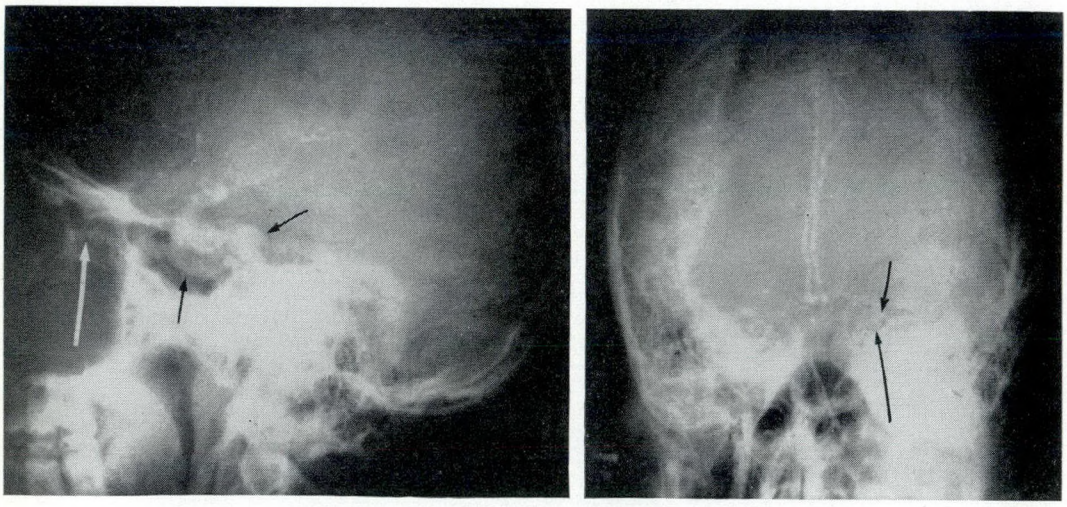

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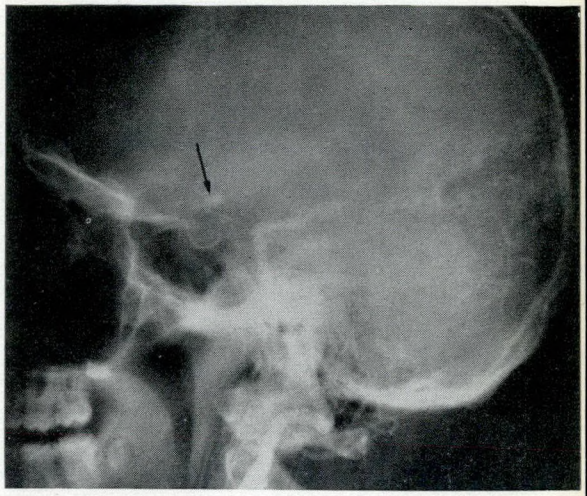
Fig. 2.—(a) Angiogram, lateral view, showing billowing clouds of contrast media in cavernous sinus spilling out into veins along the base (black arrows) as well as into large vein running into orbit (white arrow). (b) Anteroposterior view with contralateral carotid compression showing filling of fistula (between arrows) as well as irrigation of both hemispheres.

fistula. However, no catheter could be negotiated past the siphon. The passage of small polythene tubes was attempted but without success. Eventually, the intima was so badly traumatized that the lumen could no longer be identified except by a forceful jet of blood that appeared whenever the ligature on this vessel was released. Successively higher openings had been made and eventually nothing further could be introduced into this internal carotid; hence it was ligated and the incisions closed.

The pulsations and bruit persisted at the termination of the procedure but to our surprise the following day there was no bruit and no pulsation (Figs. 3 and 4). The boy remains without symptoms or signs to date, some two years later. It is assumed that our efforts to pass the various catheters and polythene tubes created some loose strips of intima which floated into the opening and acted as a flap valve, or else that the traumatized intima initiated a thrombus which propagated upwards, closing the fistula.



c

Fig. 2(c).—Vertebral angiogram showing some filling of carotid distal to shunt (arrow) but no filling of shunt.

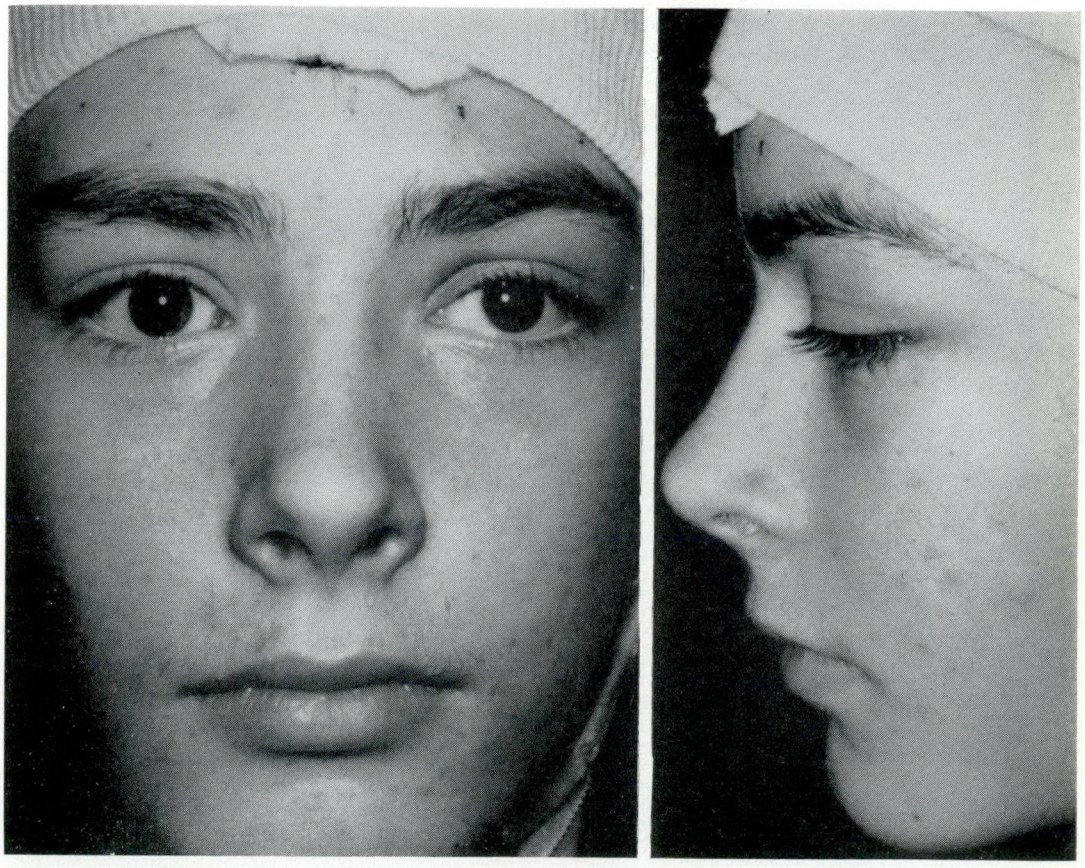


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Fig. 3.—Views of patient, postoperatively.

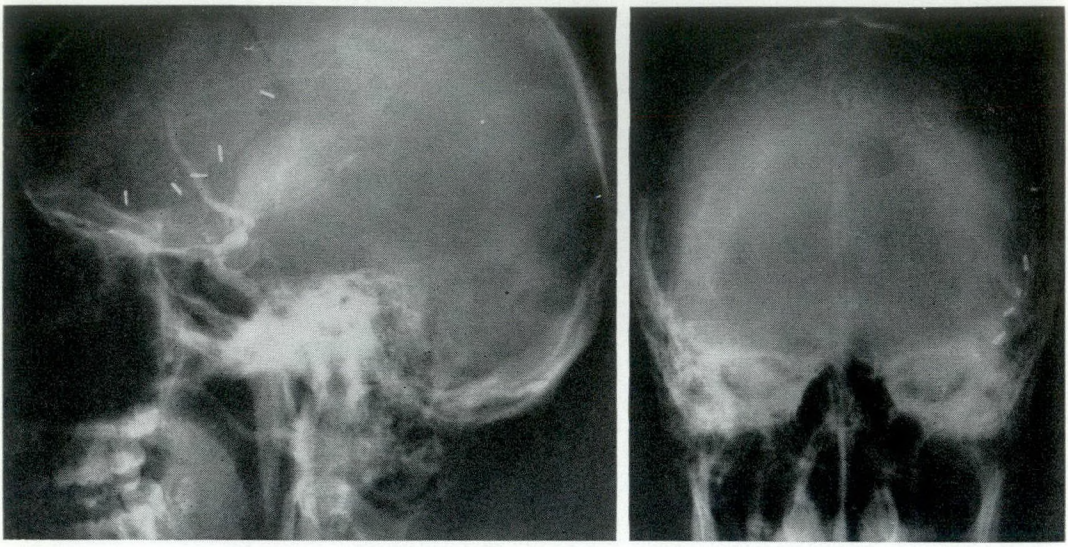

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Fig. 4.—(a) Postoperative right carotid angiogram showing no filling of shunt. Compare sellar detail with postoperative lateral views. (b) Postoperative anteroposterior angiogram showing filling of both hemispheres. Compare parasellar detail on operative side with Fig. 2c.

It is quite evident that this form of therapy should never be attempted unless beforehand one has been able to satisfactorily clip the intracranial segment of the internal carotid as it comes through the dura. It would probably be quite safe to attempt such occlusion without the additional ligation of the ophthalmic artery. (This last procedure is rather difficult in most patients owing to the overlying optic nerve.) It is also evident that any efforts at traumatizing the intima should be carried out with great caution. Perforation of the carotid artery beyond the operative field would be disastrous.

Summary

A case of a traumatic carotid cavernous fistula is presented which was fortuitously cured after trapping of the fistula and traumatization of the intima of the trapped segment of the internal carotid artery. Operation in this instance was performed within one month of the initiating trauma. No evidence of any abnormal connections to the internal carotid artery was found; this demonstrates that a voluminous collateral circulation is available to this intracranial segment of the internal carotid artery. This collateral circulation has been studied further and will be discussed in a subsequent publication.

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Résumé

Histoire d'un cas. Un enfant de 12 ans fait une chute d'un tracteur en marche et se contusionne violemment la région orbitaire gauche.

Immédiatement survient une cécité de l'œil gauche. Il est examiné très peu après. L'examen ophtalmologique montre un fond d'œil normal, mais le réflexe pupillaire à la lumière est aboli. De plus, un bruit particulier est facilement audible dans cette région, synchrone avec les pulsations cardiaques. Une angiographie permet de constater un court-circuit entre l'artère carotide interne gauche et le sinus caverneux. Environ un mois plus tard, l'enfant fut revu pour apparition d'une exophtalmie pulsatile: l'examen angiographique est peu modifié. Il fut décidé d'intervenir; on pratique la ligature de la carotide interne intracrânienne, au-dessus de l'apophyse clinoïde, de l'artère ophtalmique et enfin de la carotide interne dans le cou. Malgré cela, les pulsations et le souffle persistent; bien évidemment il s'était dévoloppé une circulation collatérale importante. On pratiqua divers tentatives de cathétérisation de la carotide interne, sans succès. On fut obligé de laisser les choses telles quelles, mais fort heureusement, le lendemain de l'opération, le souffle disparut spontanément; les auteurs pensent que ce résultat est dû à leurs tentatives de cathétérisation qui ont peut-être provoqué l'arrachement d'une partie de l'intima, et que ces débris ont initié la formation d'un thrombus qui a finalement fermé une fistule qui n'avait pu être reconnue.

ADENOCARCINOMA IN SUPERNUMERARY KIDNEY*

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THE supernumerary kidney is considered the most uncommon of all the genitourinary anomalies. Carlson,[1] in 1950, collected 51 such cases, and one additional instance has since been reported.[5] Carcinoma arising in a supernumerary kidney is therefore a very unusual circumstance and only one case has been reported, in 1944 by Exley and Hotchkiss.[2]

A supernumerary kidney is an extra (additional) renal organ, with a separate capsule and blood supply, unattached or loosely attached by connective tissue to the regular kidney on that side. This anomaly arises either on the basis of the development of two ureteric buds arising from the caudal end of the mesonephric duct, or the premature splitting of the ureteric bud with subsequent splitting of the metanephrogenic blastema, so that each bud is capped separately with renal tissue. The two metanephrogenic bodies then undergo the normal developmental process of growth, ascent and rotation, with the formation of two kidneys on that side.[6] The kidneys have occasionally been found to be of equal size and development, but as a rule the supernumerary organ is smaller and functionally deficient. The majority of supernumerary kidneys have been found to be situated inferior to the regular kidney on the involved side, with a small number being situated superiorly. A distinct ureter may be a part of the accessory organ, or the excretory apparatus may consist of a primitive remnant which terminates blindly, drains into the regular ureter on that side, or directly into the bladder through an ectopic orifice.

There are no characteristic features associated with this anomaly, except for the frequency with which secondary involvement by varied pathology occurs, the least common of which is carcinoma.

CASE REPORT

A 66-year-old white woman of Portuguese descent was admitted to the Vancouver General Hospital on March 3, 1961, for investigation of symptoms associated with intermittent left flank pain of one year's duration. Her physician had found a tender abdominal mass on routine examination. Her past history included 11 normal pregnancies, malaria and infectious jaundice, the latter while she was a resident of Shanghai, China, some 25 years previously. Functional enquiry elicited symptoms of fatty food intolerance of several years' duration, and very indefinite genitourinary symptoms, which consisted of nocturia and slight stress incontinence. Her weight had remained stationary for years.

On examination she was found to be a dark-complexioned obese woman of stated age, in no apparent distress. Examination of

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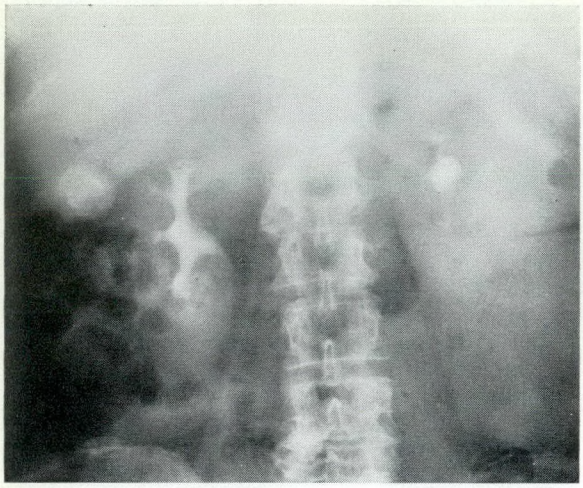

Fig. 1.—Preoperative intravenous pyelogram showing bilateral excretory function and a soft tissue mass below the left kidney.

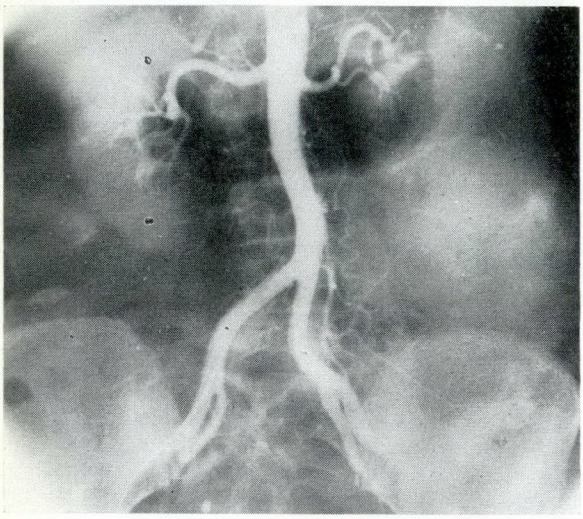

Fig. 2.—Percutaneous transfemoral aortogram outlining renal arteries and demonstrating arterial supply to mass.

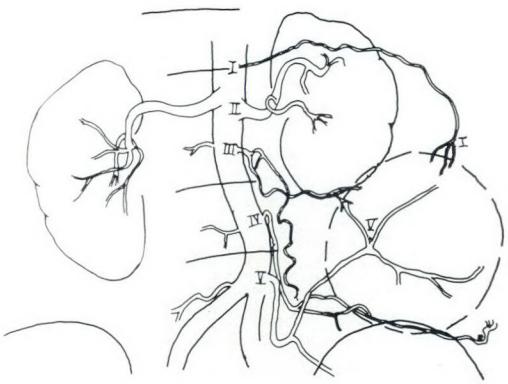

Fig. 3.—Schematic outline of aortogram in Fig. 2. I. Subcostal artery, supplying superior portion of mass. II. Renal arteries. III & IV. Lumbar arteries, main blood supply to mass. V. Inferior mesenteric artery displaced anteriorly and medially.

the heart and lungs showed no abnormality aside from an elevation of her blood pressure, which was recorded at a pressure of 170 mm. systolic and 90 mm. Hg diastolic. The mass could be palpated in the left upper abdomen and measured 15 x 10 cm., extending from the costal margin to the level of the umbilicus, and laterally into the left loin. The mass was tender on palpation, the outer surface was quite regular and ballottement was successful. On pelvic examination, a 6 to 8 cm. ovoid mass was found in the left adnexal area, and some irregular enlargement of the uterus was demonstrated as well.

Laboratory examination of the urine showed a few granular casts and occasional red blood cells, with occasional white blood cells. Blood studies showed a hemoglobin of 12.2 g. and a total leukocyte count of 9700/c.mm. with a normal differential. Erythrocyte sedimentation rate was 111 per hr. (Westergren). Further blood studies were within normal limits.

Preliminary radiological investigation showed a large soft tissue mass, situated below and distinctly separate from the left kidney. A radiopaque gallbladder calculus was seen in the right upper abdomen. Examination of the chest was unremarkable. Further radiological examination included stomach, duodenum and large bowel studies, and no abnormalities were reported, apart from a persisting soft tissue mass in the left abdomen. An intravenous pyelogram with diatrizoate sodium (Hypaque) was reported as showing good excretory function bilaterally, and slight displacement of the left kidney medially (Fig. 1). A percutaneous transfemoral aortogram was then performed using Hypaque, and this demonstrated normal renal arteries with an arterial supply to the mass, arising from the subcostal and lumbar arteries. The vascular pattern suggested a benign lesion, and was so reported by the radiologist (Figs. 2 and 3).

An exploratory laparotomy was performed on March 21, 1961. A transverse incision was made at the level of the umbilicus, and the mass was approached initially in the extraperitoneal plane. The incision was later extended, after assessment of the mass, to a combined intraperitoneal and extraperitoneal exposure. It was then found that a tumour was lying adjacent to but distinctly separate from the lower pole of the kidney. A narrow pedicle was isolated medially, and the mass was removed merely by shelling it out from the adjacent structures; this was done with relative ease. Encapsulation appeared to be complete, and the outer surface was smooth. The mass

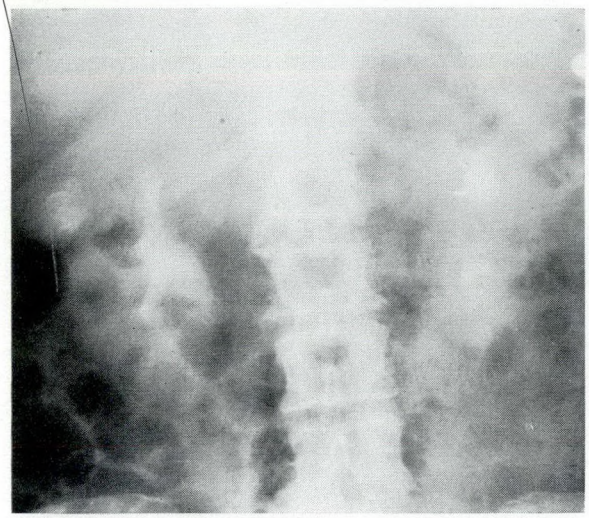

Fig. 4.—Postoperative intravenous pyelogram showing bilateral excretory function.

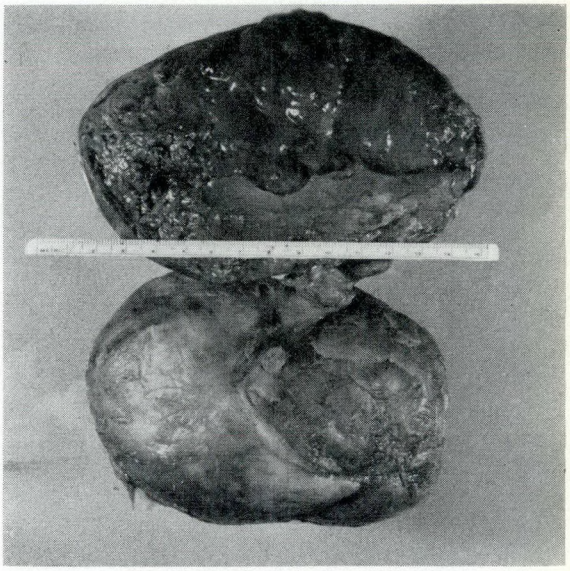

Fig. 5.—Gross appearance of mass showing external and cut surfaces.

measured 15 x 10 x 8 cm. The left ureter was visualized after removal of the mass and grossly appeared to be completely normal. No ureteric structures could be identified on the surface of the tumour mass, and no attached remnants could be seen along the course of the ureter.

Arising in the right pelvis was a large multiloculated cystic structure of ovarian origin, measuring 8 x 6 cm.; this was subsequently removed along with a similar-appearing structure of smaller size arising from the left adnexal region. The remainder of the abdominal organs were normal, apart from a palpable calculus in the gallbladder. Retroperitoneal drains were inserted and the wound was then closed in a routine manner. The patient's postoperative course was uneventful.

Repeat intravenous pyelographic examination showed satisfactory bilateral function; the ureters were unchanged when compared with the previous studies (Fig. 4).

Pathological examination of the tumour revealed it to be a soft encapsulated structure, which on cross-section was seen to have a fibrous capsule 2 to 6 mm. in thickness. The central part was friable, of yellowish-grey colour with hemorrhagic areas and presented an overall variegated appearance (Fig. 5).

On microscopical examination large areas of hemorrhagic necrotic tissue were noted, and peripherally there were adenomatous neoplastic agglomerations. Distinct glomeruli and tubules were present (Fig. 6). The appearance was consistent with that of a "clear-cell" adenocarcinoma with a large central area of necrosis and hemorrhage (Fig. 7). The cystic structures removed from the adnexae were benign serous cystadenomata.

Fig. 6.—Photomicrograph of peripheral area of mass; the glomeruli and tubules are relatively normal in appearance (x 125).

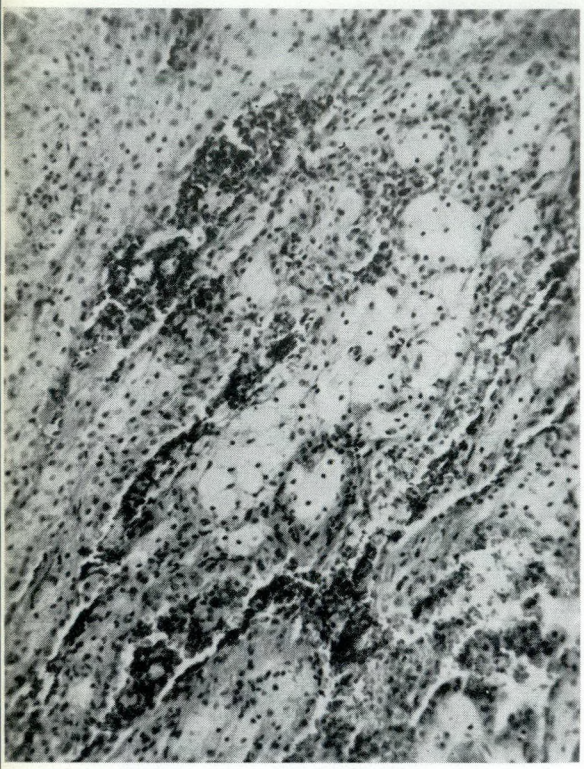

Fig. 7.—Photomicrograph of representative area of large clear tumour cells arranged in tubular and papillary formation (x 160).

Discussion

This case of carcinoma arising in a supernumerary kidney is presented as a rare occurrence. The preoperative diagnosis was a retroperitoneal tumour. The operative findings were a large encapsulated structure unattached to the left kidney or ureter, with a medial pedicle consisting of two small arteries and veins. No ureter or pelvis was found associated with the tumour, and it is unlikely that the glomeruli and tubules in the peripheral parts of the tumour, which were of normal appearance on histological examination, were functioning.

Summary

This is the second reported case of adenocarcinoma of the kidney arising in a supernumerary organ. A brief review of the literature and an outline of the embryology of this anomaly is presented. The patient remains well to date (February 1963).

The authors wish to thank Dr. H. K. Fidler, Department of Pathology, Vancouver General Hospital, for his assistance in the preparation of this report. They are also indebted to Dr. David Garrow, Department of Radiology, and Dr. Leon Komar, Department of General Practice, for their help.

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Résumé

Histoire d'un cas. Une femme de 66 ans est admise au "Vancouver General Hospital" en mars 1961 pour une douleur intermittente du flanc gauche remontant à un an en arrière. L'examen physique permet la palpation d'une tuméfaction dans le quadrant supérieur gauche de l'abdomen, mesurant environ 15 x 10 cm., de consistance molle, de surface irrégulière, non fixée. L'analyse d'urine montre la présence d'un peu de sang. Un cliché radiologique de l'abdomen à vide met en évidence cette même tuméfaction ainsi qu'un calcul biliaire. Une laparotomie exploratrice est faite: après incision transverse, on atteint la tumeur par voie rétropéritonéale. Cette tumeur est située à proximité immédiate du pôle inférieur du rein, mais bien séparée de ce dernier. L'ablation est pratiquée sans difficultés majeures, après ligature d'un pédicule vasculaire bien individualisé. L'examen microscopique montre de larges plages de tissu hémorragique et nécrotique, et en périphérie des agglomérations néoplasiques adénomateuses; de plus, il existe des tubes et des glomérules rénaux. Il s'agissait donc d'un adéno-carcinome développé dans un rein surnuméraire, éventualité particulièrement rare. On ne trouve qu'un seul cas de ce genre décrit dans la littérature. Il faut noter que dans le cas présenté ici, le rein supplémentaire n'avait pas de relation avec le rein normal et n'avait pas non plus de bassinet ou d'uretère. Les auteurs résument l'embryologie de cette question.

EXPERIMENTAL SURGERY

STUDIES OF THE FIRST SUCCESSFUL DELIVERY OF AN UNBORN LAMB AFTER 40 MINUTES IN THE ARTIFICIAL PLACENTA*

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THE members of the Division of Thoracic and Cardiovascular Surgery of the University of Alberta Hospital became interested in the development of an artificial placenta[1, 2] as a means of providing a controlled state for studies in long-term perfusion. The possibility was entertained of using such a device for long-term support for premature infants and those suffering from respiratory distress syndrome of the newborn.[3]

We chose the lamb as the experimental animal and in doing so were guided by the excellent physiological studies of Dawes,[4] Barcroft and Barron[5] and Barclay, Franklin and Prichard.[6] We were also stimulated by the work of Westin, Nyberg and Enhörning,[7] of Stockholm, who in 1950 kept seven previable human fetuses alive for periods of up to 12 hours with no attempt at initiation of voluntary respiration. Harned *et al.*[8] referred in a discussion to having supported a newborn lamb on the pump oxygenator for as long as an hour but no details of their work were published in the literature.

The purpose of this report is to describe in detail the survival of the first lamb sustained by means of the artificial placenta and to compare the studies made on this animal with those of lambs that were carried under similar conditions but which for one reason or another failed to survive in the atmosphere.

METHODS

Ten animals varying in length of gestation from between 80 to 134 days (normal for the Suffolk ewe, 144 to 147 days) were delivered by Cesarean section from eight Suffolk ewes. Their weights varied between 0.6 to 4.0 kg. The ewes were anesthetized with pentobarbital (Nembutal) intravenously or by epidural lidocaine (Xylocaine).

The hind quarter of the lamb was delivered first to permit cutdown and cannulation of the femoral artery with polythene No. 60 catheters so that the pressure and biochemical studies could be carried out. An umbilical vein and both umbilical arteries were dissected out with fine scissors and two No. 4 silk sutures were placed around each vessel at either end of a 2.5 cm. length cleared by the dissection; this was approximately 5 cm. from the skin surface. An umbilical vein catheter was inserted to a depth of 5 cm. from the skin and the umbilical arteries were cannulated until resistance to further passage was felt; this was usually close to the internal iliac vessels. If the animal's condition deteriorated, the second umbilical artery was cannulated after the perfusion had been started. At the time of cannulation, heparin was injected into the umbilical vein in the amounts of 2 mg./kg. body weight of the fetus.

Polyvinyl Bardic catheters of appropriate sizes were held by a stainless steel holding device (Fig. 1). Catheters were attached

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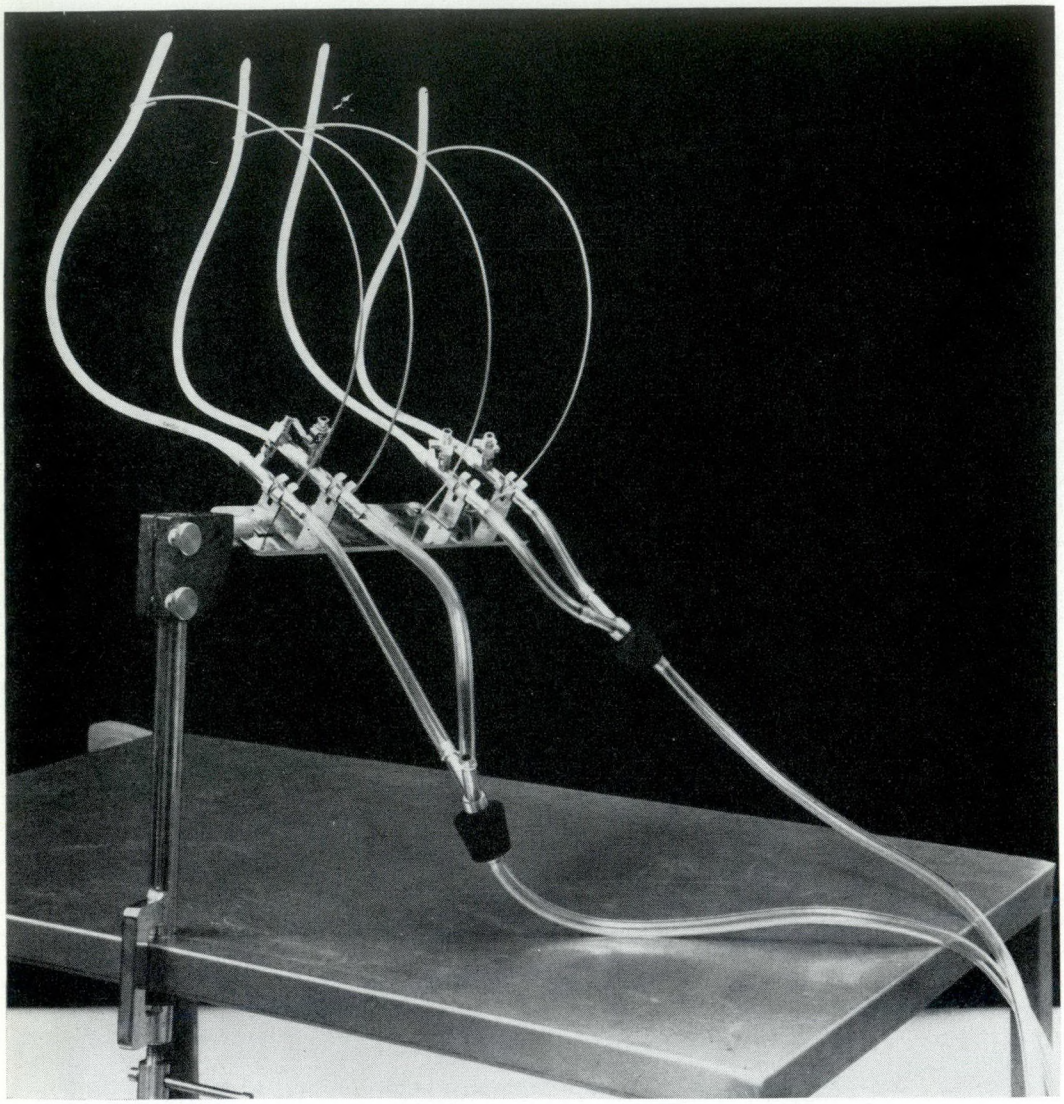

Fig. 1.—The device for holding the umbilical cannulae.

to the perfusion apparatus, which had been primed with 1400 to 1500 c.c. of donor blood taken under local anesthesia from ewes other than the mother. (The experimental set-up of equipment used in this procedure is demonstrated in Fig. 2.) The fetus was then transferred to the placental chamber (Fig. 3) without exposing the the head to the atmosphere, thus preventing initiation of respiration. The animal was immersed in artificial amniotic fluid of 5% glucose and normal saline at temperatures between 35° to 39° C. The animal was permitted to pump desaturated blood via the umbilical artery into the first reservoir, termed the umbilical artery reservoir. From here the blood passed through an oxygenator which consisted of a rotating disc oxygenator in eight of the animals and a membrane oxygenator of special design in the remaining two. Blood was then passed through a bubble trap and heat exchanger to another reservoir, termed the umbilical venous reservoir, and returned from here to the animal by gravity. The height of both reservoirs could be changed according to the flow of the individual animal being perfused.

The umbilical vein and artery blood was sampled before perfusion and at 10, 20, 30

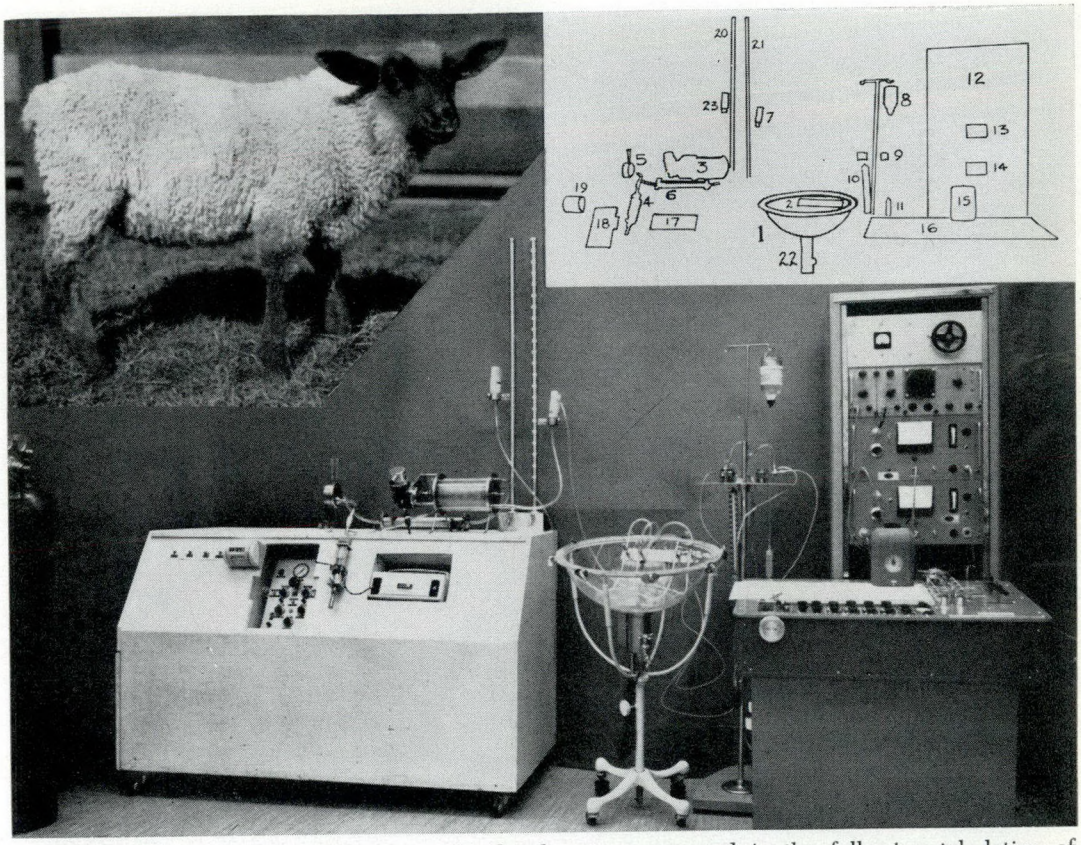

Fig. 2.—The experimental set-up. The details are enumerated in the following tabulation of component parts. 1. Placental chamber. 2. Holding device for cannulae. 3. Rotating disc oxygenator. 4. Davol ventricle pump. 5. Stainless steel filter. 6. Heat exchanger. 7. Umbilical vein reservoir. 8. Saline strain gauge. 9. Pressure strain gauges. 10. Mercury manometer. 11. Intravenous tubing. 12. Twin-beam oscilloscope. 13. Umbilical artery electromagnetic flow meter. 14. Umbilical vein electromagnetic flow meter. 15. Registering time clock. 16. Eight-channel recorder. 17. Tele-thermometer. 18. Davol pump control. 19. Elapsed-time clock. 20. Umbilical arterial reservoir, adjustable control column. 21. Umbilical venous reservoir, adjustable control column. 22. Artificial amniotic fluid heat control. 23. Umbilical artery reservoir. (Inset) Experiment 304. First successful delivery from 40 minutes in artificial placenta, three months later.

and 40 minutes, and again when the animal was returned to the atmosphere. Oxygen saturations in the blood were measured by the Thomas-Van Slyke manometric method and the Beckman Spinco Gas Analyzer Model 160. Carbon dioxide content was measured by the Natelson Microgasometer and the pH by the Photovolt pH meter. Hematocrit was measured by the Winthrop method and the hemoglobin, plasma hemoglobin, blood urea and blood sugar estimations were made on the Klett colorimeter.

Electrocardiogram, electroencephalogram, umbilical vein and artery pressures were measured by means of Statham pressure transducers and recorded on an eight-channel Gilson Macropolygraph. Umbilical artery and vein flow rates were measured on a Medicon four-channel electromagnetic flow meter.

Results

Of the 10 animals studied, it was possible to obtain satisfactory perfusion in seven for periods of longer than 40 minutes and three of these animals were delivered to the atmosphere. Of the three delivered to the atmosphere, one died of hemorrhage at eight hours, one was sacrificed for pathological study and one is still surviving and is now four months of age and apparently thriving (Fig. 2—inset). Fig. 4 shows the results in terms of the

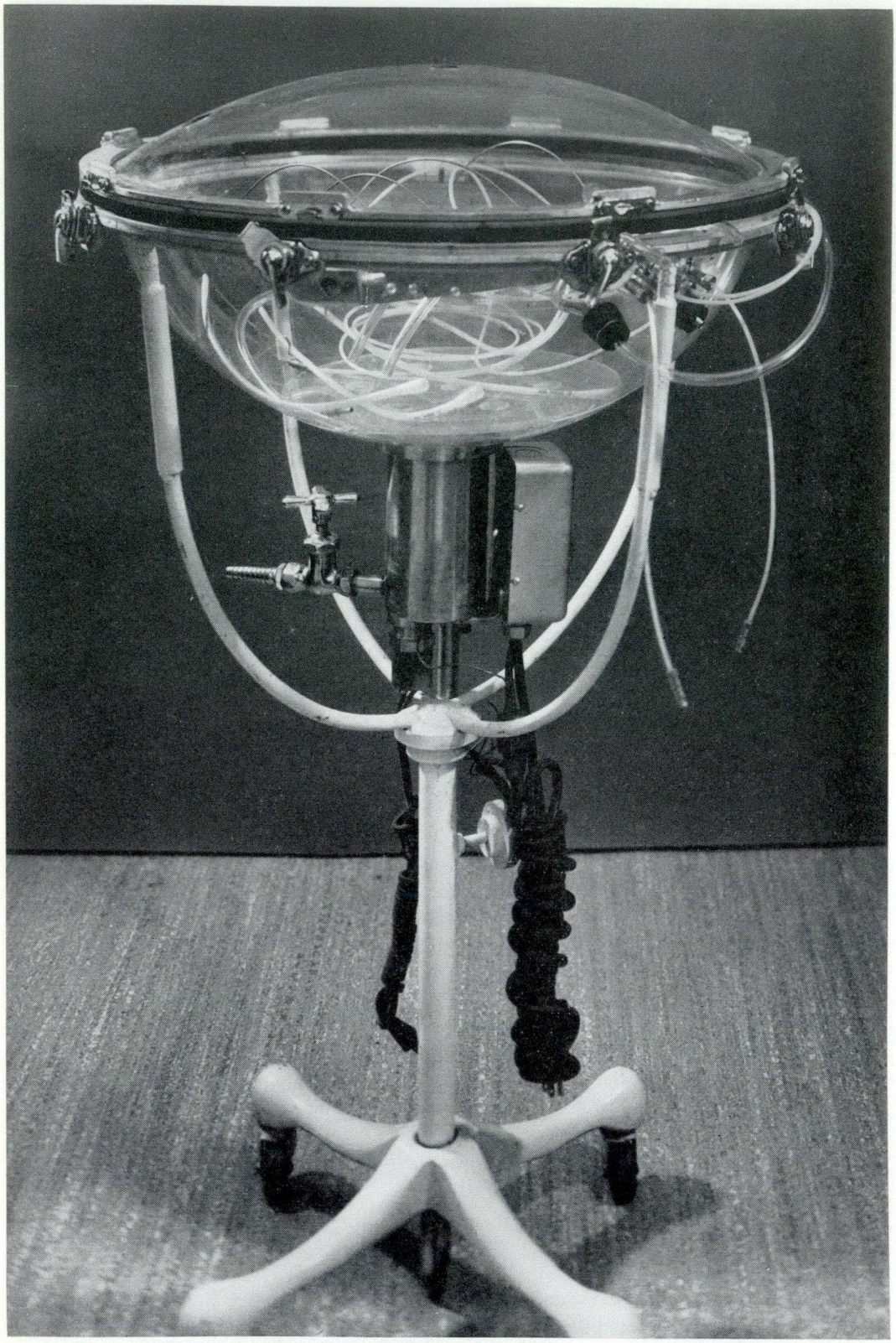

Fig. 3.—A close-up of the placental chamber. The heat control may be seen just under the chamber.

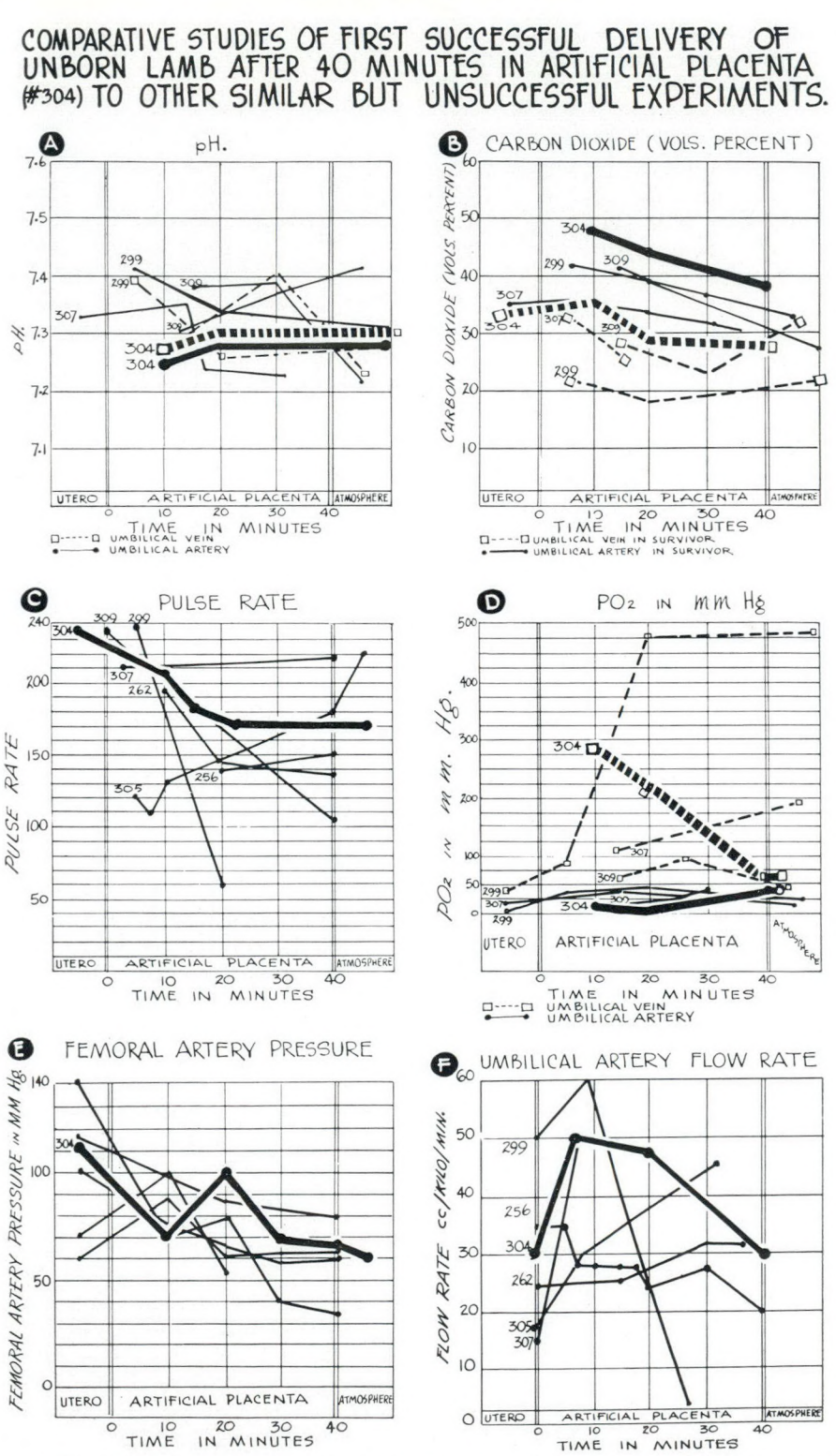

Fig. 4.—Various parameters in the long-term survivor are compared with those of lambs under similar conditions who failed to survive in the atmosphere.

various parameters measured in the first long-term survivor of 40 minutes perfusion compared with those of other animals in similar but unsuccessful experiments.

The flow rates varied between 13 to 60 c.c./kg./min. In all but the survivor, the flow rates at times fell to less than 30 c.c./kg./min. The pulse rate varied from 120 to 240 per minute during the early perfusion; if a major decrease in pulse rate occurred, this heralded the demise of the animals in all instances. In the three animals delivered to the atmosphere, the pulse rate did not fall below 160 per minute. In the long-term survivor, Experiment 304, the pulse rate was 230 per minute at the beginning of the experiment and was 170 on return to the atmosphere. The oxygen tension of the umbilical artery remained below 50 mm. Hg in all animals at all times. The wide variation in pO_2 values in the umbilical vein reflected the varying flow rates to the oxygenator (Fig. 4d).

A comparison of the carbon dioxide content of venous and arterial blood of the survivors and non-survivors is shown in Fig. 4b. There was a gradual fall in carbon dioxide content of the umbilical artery blood in all animals. The pH showed a tendency to fall in the non-survivors but remained fairly constant in the long-term survivor (Fig. 4a). Blood glucose values varied between 74 and 80 mg. % in all of the animals studied.

The electrocardiogram demonstrated anoxic changes when a fall in pulse rate was associated with a widening of the QRS complex, T-wave changes and decrease in voltage. When the animal was submerged, technical difficulty was experienced in obtaining a good tracing routinely; however, the heart rate, if not the characteristics of the tracing itself, could be determined at all times.

The electroencephalogram was extremely difficult to obtain in submerged animals owing to the failure, even with our deeply embedded electrodes, to prevent conduction of currents in the fluid medium.

The femoral artery systolic pressures *in utero* varied between 60 to 140 mm. Hg in the six animals studied (Fig. 4e). As the perfusion progressed the femoral artery pressure tended to fall. In the surviving animal it remained above 60 mm. Hg at all times; this was the value recorded in the animals who survived under atmospheric conditions.

Discussion

In an early study we were able to keep lambs alive in the artificial placenta for periods of longer than eight hours; the longest period of survival was 19 hours. However, as a result of pathological examination, it was concluded that none of these animals was capable of surviving in the atmosphere because of lung congestion and hemorrhage.

There were probably multiple reasons for the failure of these animals to survive under these conditions. However, cannulation, which took too long to accomplish, and inadequate perfusion rates appear to be important contributions to failure in this particular experiment. On the average, the flow rate of the survivor was appreciably higher than those of the other animals who were similar in other respects but failed to survive in the atmosphere.

Adams and Lind[9] found flow rates in the human fetus to change from 60 c.c./kg./min. before birth to as high as 273 c.c./kg./min. just after birth. Dawes[4] demonstrated umbilical flow rates of 100 to 180 c.c./kg./min. in six premature lambs. We were unable to reach this level by our present techniques but current developments in cannulation have improved our flow rates. Undoubtedly, this type of cannulation, which is dependent on the umbilical arteries, is subject to very small flow owing to spasm resulting from the stimulation of the umbilical vessels; direct cannulation of the internal iliac artery may well obviate this diminution of flow. In any human application of similar perfusion techniques, the internal iliac artery is undoubtedly the vessel of choice and is used in our present method of cannulation on the arterial side. Dawes also showed that in a heart-lung preparation of the lamb, a decrease in the size of the ductus arteriosus occurred when the arterial oxygen saturation rose above 76% or went to extremely low levels. In none of our animals did the peripheral oxygen tension rise

above 50 mm. Hg. Dawes calculated the mean cardiac output of the fetal lamb to be 115 c.c./kg./min. just before birth and found that it rapidly rose to 325 c.c./kg./min. after birth. These values indicate that higher flow rates must be obtained if perfusion is to be physiologically stable for a long period. This conclusion is probably the most significant one reached as a result of our studies on such preparations to date.

In preparation for the clinical application of such long-term perfusion, we have used a veno-venous perfusion in dogs for periods up to 12 hours; routine survival has been obtained after four hours of this form of perfusion when the tracheobronchial tree has been obstructed or the animal immersed in fluid. At the present time, further studies are under way into techniques of improving flow rates in this method of perfusion. The possibility is also being investigated of substituting a more easily controlled form of membrane oxygenator which appears to provide long-term perfusion without causing the disturbances in formed blood elements so common in the blood-air (or oxygen) interface-type oxygenators.

Summary

Twenty-eight lambs have been transferred by Cesarean section to the artificial placenta and perfused with oxygenated blood via the umbilical vessels for up to 19 hours while submerged in artificial amniotic fluid. An attempt was made to deliver 10 of these animals to atmospheric conditions. Three of these animals survived for periods of longer than six hours and one animal is a long-term survivor at four months.

Changes in pH, CO_2 content, oxygen tension, blood sugar, flow rates and blood urea nitrogen in those animals that failed to survive were compared with those values in the single long-term survivor. These laboratory determinations were also compared with information obtained under normal intrauterine conditions reported by other authors. A modification of placental circulation in which only venous cannulation is used is suggested as a possible method of support for respiratory distress in the newborn but further studies are needed to make this form of support practical in the newborn period.

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Résumé

Les auteurs présentent ici les résultats d'une série d'expériences faites dans le département de chirurgie thoracique et cardio-vasculaire du "University of Alberta Hospital" sur le placenta artificiel. Ces expériences ont porté sur dix agneaux dont le temps de gestation variait entre 80 et 134 jours (gestation normale: 145 jours environ), et le poids entre 0.6 et 4.0 kg. Ces animaux furent extraits par césarienne. Une artère fémorale, la veine ombilicale et les artères ombilicales furent soigneusement disséquées et cathétérisées avec des tubes en polythène; on contrôlait la coagulation sanguine par des injections d'héparine. Ces cathéters, furent reliés à un système de circulation artificielle dans lequel on avait préalablement placé un volume de 1.500 ml. de sang provenant d'une brebis donneuse autre que la mère. Le fœtus était alors transféré dans une chambre placentaire et plongé dans un liquide amniotique artificiel composé essentiellement de solution physiologique additionnées de 5% de glucose. La température était maintenue constante entre 35° C. et 39° C. L'oxygénateur utilisé était du type

à disque rotatif dans huit cas et à membrane dans les deux derniers. Des contrôles biochimiques furent faits sur le sang circulant et l'on enregistra l'électrocardiogramme, l'électroencéphalogramme et la pression sanguine dans les artères et les veines ombilicales. Sur les 10 animaux qui furent soumis à ces expériences, il ne fut possible de réaliser une perfusion satisfaisante pendant plus de 40 minutes que dans seulement trois cas. Ces trois fœtus furent "accouchés" dans l'air libre; l'un d'eux mourut d'hémorragie huit heures plus tard; le second fut sacrifié pour étude anatomopathologique. Le troisième survécut parfaitement et est maintenant âgé de quatre mois, en bonne santé. Parmi les facteurs qui semblent jouer un rôle important dans les échecs qui ont été observés, il faut citer: l'opération même de la canulation qui prend un temps considérable, les difficultés inhérentes à la perfusion. Sur ces points, les données qui ont été obtenues par les auteurs sont discutées en détail et comparées avec les résultats d'autres auteurs.

A PRELIMINARY REPORT CONCERNING THE USE OF TRIS BUFFER (THAM) IN THE TREATMENT OF HEMORRHAGIC HYPOTENSION IN THE DOG*

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An investigation into the use of an organic buffer (THAM)¶ in the treatment of severe standardized hemorrhagic hypotension was carried out during 1961.

Secondary or surgical shock is a complex clinical state which may be reproduced in the experimental animal in many ways.[14] A standardized method of producing hemorrhagic hypotension has gradually been evolved in the surgical laboratories at the University of Western Ontario Medical School between 1951 and 1955 by a succession of surgical residents investigating the efficacy of various replacement fluids in the treatment of hemorrhagic hypotension[3, 6, 7] (see Table I).

It was concluded from their work, first, that 70 minutes of controlled hypotension in the dog gives a reproducibly severe, yet reversible degree of shock, and secondly, that dextran is no more effective than saline in the treatment of shock due to blood loss.

Severe hemorrhagic shock causes significant physiological and biochemical abnormalities. The impairment of circulation is caused by a decrease in blood volume and is aggravated by preferential shunting and sludging in capillaries. The secondary anoxia leads to disturbances in tissue metabolism and acidosis. The acidosis is related to CO_2 retention and to anaerobic metabolism with resultant accumulation of

TABLE I.—Results of Blood-Loss Replacement Studies

Group	*Treatment*	*No. of animals treated*	*Results*	*% survival*
	A. *McFarlane*[6]			
I	None	12	10 died	16
II	Blood	12	1 died	92
III	Dextran	12	6 died	50
IV	Normal saline	12	6 died	50
	B. *McLarty and McLachlin*[7]*			
I	None	20	18 died	10
II	Blood	20	3 died	85
III	Dextran	20	10 died	50
IV	Normal saline	20	11 died	45
	C. *Carroll and McLachlin*[3]			
I	None	10	10 died	0
II	Blood	9	3 died	66
III	Double-volume saline†	20	10 died	50
IV	Salt-free dextran in 5% glucose	10	9 died	10
	D. *Present study*			
I	None	13	13 died	0
IIa	5% glucose in distilled water	13	12 died	8
IIb	THAM in 5% glucose and distilled water	12	1 died	92

*A standardized form of trauma was added in this series of experiments.
†A volume of normal saline equal to twice the mean volume of blood in the bleeding reservoir.

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¶Abbott Laboratories Ltd.

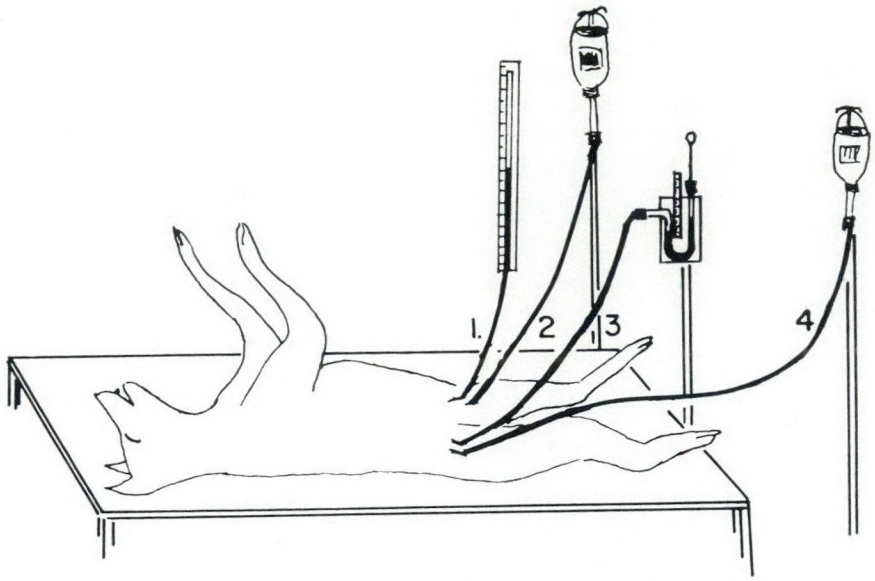

Fig. 1.—Method of controlled hypotension. (1) Saline manometer for central venous pressure. (2) Blood reservoir connected to femoral artery. (3) Mercury manometer for mean arterial pressure. (4) Intravenous infusion.

pyruvic and lactic acids. The acidosis presumably is intracellular as well as extracellular, and is compounded by renal failure and defective handling by the liver of pyruvic and lactic acids.

Effective therapy for severe hemorrhagic hypotension requires restoration of blood volume and correction of the metabolic derangements as well; and important among these derangements is intracellular and extracellular acidosis. The implementation of this concept in other hands has led to the use of sodium bicarbonate (the only previously acceptable alkaline substance) without much success. It therefore seemed worthwhile to investigate the usefulness of the new amine buffer THAM for this purpose. This organic buffer has already been shown to be effective in controlling both respiratory and metabolic acidosis produced or occurring by various means.[9, 17-19]

This study was commenced in June 1961, and it was not until October 1961 that we learned that other investigators[4] had used THAM in combination with bicarbonate or oxygen in the treatment of hemorrhagic hypotension.

Materials and Method

Forty-five healthy adult mongrel dogs were used in this study and divided into three groups. As necessary, each was anesthetized with pentobarbital sodium 30 mg./kg. and the trachea was intubated. The groins were shaved and the femoral vessels on both sides were cannulated. The mean arterial pressure was recorded with a mercury manometer after the animal was given 1.5 mg. of heparin per kg. of body weight.

The animal was bled from the opposite femoral artery into a reservoir which was adjusted to the height necessary to main-

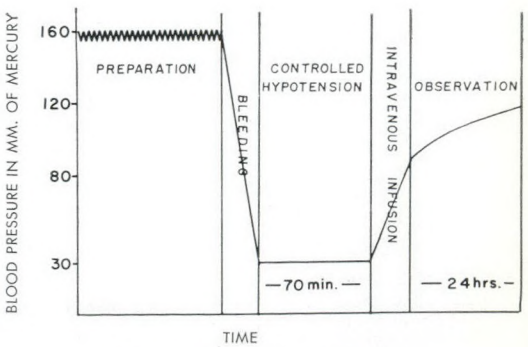

Fig. 2.—Plan of experiment.

tain the mean arterial pressure at 30 mm. Hg. Arterial samples were taken at fixed intervals for biochemical studies. The solution to be tested was injected into a femoral vein. Central venous pressure was measured by a polyethylene catheter inserted into the inferior vena cava.

The dogs were bled rapidly (in 3 to 5 minutes) until the mean pressure reached 30 mm. Hg. This pressure was then maintained for 70 minutes.

Group I

Fifteen animals were subjected to this procedure and no treatment was administered subsequently.

Group II

Thirty animals were then subjected (by pairs) to this method of producing hemorrhagic hypotension. In one of each pair the blood loss (as measured by the greatest amount in the reservoir during the 70 minutes of the hypotension) was replaced by 5% glucose in distilled water (Group IIa). The other animal was given tris buffer (THAM) in a dose of 400 mg./kg. body weight diluted in 5% glucose in distilled water (Group IIb). The volume of the latter was again determined by the blood loss during the 70 minutes of hypotension.

Observations: Group I.—Two of the 15 animals in the untreated group failed to survive the 70 minutes of hypotension. None of the remaining 13 animals survived for 24 hours.

Observations: Group IIa.—Of the 15 animals in this group, two failed to survive 70 minutes of hypotension. Twelve died within 24 hours, and one survived more than 24 hours.

Observations: Group IIb.—Of the 15 animals in this subgroup, three failed to survive the 70 minutes of hypotension. The remaining 12 animals were given THAM (400 mg./kg.) in 5% glucose in distilled water. Eleven of the 12 recovered and survived indefinitely and one animal died after three hours.

On the basis of Mainland's tables for statistical analysis,[8] the difference between the group treated with glucose in distilled water and the group treated with THAM in glucose solution is highly significant. ($P < .001$).

Discussion

This method of producing experimental hypovolemic "shock" may be questioned, but after an accumulated experience with it in approximately 350 dogs, we feel confident that 70 minutes of controlled hypotension produced in this way gives a reproducibly severe insult which is fatal to the dog unless effectively treated. The severity of the method is emphasized by the fact that seven of the 45 animals in the present study failed to survive 70 minutes of hypotension. Furthermore, it should be noted that the mean volume of blood in the reservoir was 53 c.c./kg. of body weight; that is, more than half of the animal's blood volume. The shock in these dogs was not irreversible because blood replacement allowed most of them to survive (Table Ia). It has been demonstrated in our laboratory that restoration of blood volume alone by the intravenous injection of dextran in 5% glucose will not bring about recovery from this degree of hypovolemic shock.[3] It is accepted as axiomatic that the ideal management of hypovolemic shock is the rapid replacement of lost fluid, usually by blood. Tris buffer is the best substitute presently available.

Our opinion is that survival is the best criterion for the adequacy of the treatment of shock.

THAM is an organic amine buffer, soluble in water, accepting both $H+$ and CO_2.[10] Because of its ability to penetrate cell membranes it exerts its buffering action on the intracellular component as well as on the extracellular fluids, and an important fraction of its total buffer effect takes place inside the cells.[16] A hypoglycemic effect has been observed,[12] and this is one reason why 5% glucose has been selected as the vehicle. Tris buffer in large doses is a respiratory depressant.[1, 5]

Metabolic acidosis is one feature of the shock syndrome related to failure of hepatic and renal mechanisms and to a shift of carbohydrate metabolism toward the anaerobic phase. Correction of the extracellular pH changes with sodium bicarbonate in

our laboratory and in the hands of several other investigators has not produced survival in severe shock.[14, 15]

These dogs were mildly acidotic before bleeding. Severe metabolic acidosis developed during the hypotensive period (pH 6.9-7.2) which was counteracted by the tris buffer.

Thus far, we have been unable to explain the protective effect of tris buffer in hemorrhagic hypotension.[2] The pH was restored to normal (7.35-7.45) in the THAM-treated group, but similar pH changes occur when sodium bicarbonate is given instead of THAM. However, the survival rate is not improved.[15] The blood pressure response to treatment with THAM and the response to treatment with 5% glucose in distilled water only were comparable. The pulse rate, and the central venous pressure was similar in the two groups, e.g Groups IIa and IIb. Electrolyte studies in a few experiments exhibited no significant change. Blood glucose concentrations were elevated during hypotension, but no hypoglycemic effect has been observed. Unlike Hassam and his group,[4] we have not found oxygen administration or combination with bicarbonate to be required. Tanaka, Paton and Swan[13] have observed that tris buffer has a protective effect upon the myocardium during inflow occlusion under hyperthermia. Since decreased cardiac output, perhaps from myocardial depression, is characteristic of shock, we might speculate that protection of the myocardium will explain why these dogs survived. In well-controlled experiments, Goetz, Selmonosky and State[20] have demonstrated the apparent value of THAM in preventing anuria during hypovolemic shock. This may be an additional reason for survival of these animals.[11]

Conclusions

Tris buffer in a dose of 400 mg./kg. given in 5% glucose and water is effective treatment for experimental hemorrhagic hypotension, and is the most effective blood substitute for the treatment of experimental hemorrhagic hypotension presently available. The potential value of THAM in the treatment of mass casualties may be significant.

These experiments were carried out with the technical assistance of Mr. T. Walko and Mr. G. DeYoung. The authors wish to thank Dr. Allan Ramsay for his advice and assistance. They also desire to express their gratitude to Dr. James A. Dauphinee, Professor of Pathological Chemistry, University of Toronto, for a painstaking review of this work and for helpful suggestions, many of which have been included in the revised manuscript.

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Résumé

Ce rapport présente les résultats de plusieurs séries de travaux expérimentaux effectués dans le laboratoire de chirurgie de l'école de médecine de "University of Western Ontario" sur les causes du choc chirurgical. Cette entité est un état clinique complexe qui peut être provoqué chez l'animal de beaucoup de façons. Il a été trouvé, chez le chien, qu'un état d'hypotension contrôlée pendant 70 minutes mène à l'apparition d'un choc grave, mais non irréversible; le dextran s'est révélé inefficace dans le traitement des chocs hémorragiques. La présente série d'expériences visait à étudier la valeur du tampon tris (THAM) dans la lutte contre le choc hémorragique hypotensif. A cette fin, on utilisa 45 chiens adultes anesthésiés au pentothal et intubés. On procéda à une saignée rapide provoquant une chute de tension aux environs de 30 mm. de mercure. Cette pression était maintenue pendant 70 minutes. Dans ces conditions, on répartit les animaux en trois groupes: un premier groupe ne reçut aucun traitement; le second (30 animaux) reçut un volume de solution glucosée à 5% équivalent à la quantité de sang perdue; le troisième (15 animaux) reçut du tampon tris à raison de 400 mg. par kg. de poids corporel, dilué dans une solution glucosée à 5%. Les animaux du premier groupe moururent tous. Dans le second groupe, un seul animal survécut plus de 24 heures. Dans le dernier groupe, 11 des 15 animaux se rétablirent parfaitement. Les auteurs en concluent que le tampon tris, utilisé concurremment avec une solution glucosée se révèle très efficace dans la lutte contre le choc hémorragique. Son action probable est, qu'il contrôle et réduit l'acidose respiratoire et métabolique qui accompagne ces états. Ces constatations prennent un grand intérêt lorsqu'on songe aux possibilités de traitement ainsi ouvertes en cas de catastrophes de masse.

AN EXPERIMENTAL STUDY OF PANCREATITIS FOLLOWING POLYA GASTRECTOMY*

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Pfeffer, Stasior and Hinton[8] described an original method of producing hemorrhagic pancreatitis in dogs. A segment of duodenum, 7 to 10 cm. in length into which the pancreatic ducts open, is converted into a blind loop after thoroughly irrigating the lumen with sterile saline. The common bile duct is divided and the pyloric end of the stomach anastomosed to the distal duodenum (Fig. 1). Distension of the blind duodenal loop occurred and within 11 hours hemorrhagic pancreatitis involving all the pancreas developed. They suggested that distension of the duodenal loop with secretions interfered with the blood supply to the duodenum and pancreas, resulting in hemorrhagic pancreatitis.

We anticipated that Pfeffer's procedure would provide a reliable method of producing acute hemorrhagic pancreatitis in dogs, by means of which certain enzyme studies could be undertaken. The operation was performed according to Pfeffer's original description on five dogs. Gross distension of the duodenum developed in every case with thinning of the wall and areas of necrosis at the antimesenteric border. Perforation of the duodenum through a necrotic patch occurred in two dogs, resulting in death 16 hours after operation; the other three dogs were sacrificed 20 hours after operation. Confluent hemorrhagic pancreatitis did not develop in any of the animals. Instead, the glands were very edematous and contained pinpoint hemorrhagic foci, especially in the head of the pancreas (Fig. 2). Microscopical examination confirmed the exten-

*From the Department of Surgery, University of Alberta, Edmonton, Alta. Presented at the Annual Meeting of the Royal College of Physicians and Surgeons of Canada, Toronto, January 1962.

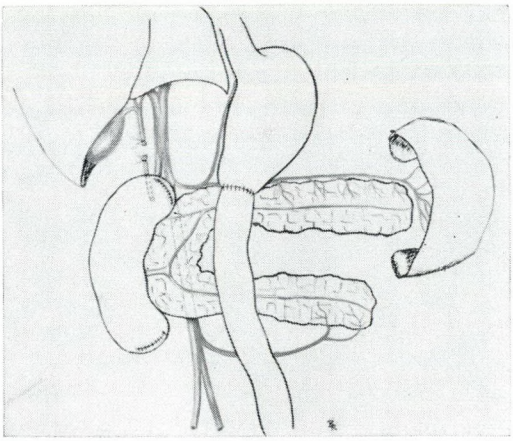

Fig. 1.—Diagram of the Pfeffer procedure.

sive edema and showed a cellular infiltration with a few polymorphs associated with scattered focal areas of coagulation necrosis (Fig. 3). Paulino-Netto and Dreiling[5] had similar difficulties in producing hemorrhagic pancreatitis using Pfeffer's method. Many of their dogs died of perforation of the isolated duodenal loop.

It seemed possible that Pfeffer's procedure might be made more efficient by combining it with a moderate insult to the pancreatic venous drainage. Therefore, in addition to making an isolated duodenal loop and dividing the common bile duct, the superior pancreaticoduodenal vein was also tied (Fig. 4). Pfeffer's operation combined with ligature of the superior pancreaticoduodenal vein was performed upon 10

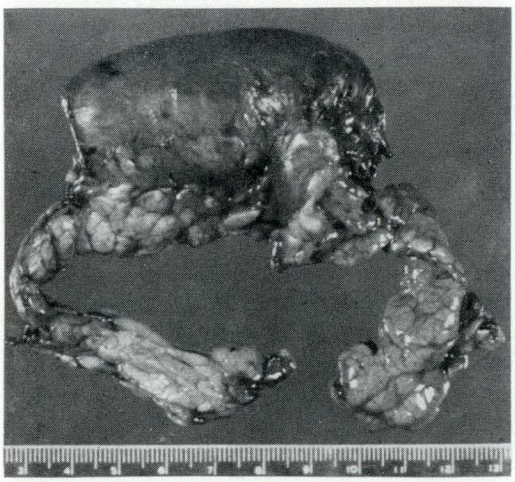

Fig. 2.—Gross changes in the pancreas 20 hours after the Pfeffer procedure.

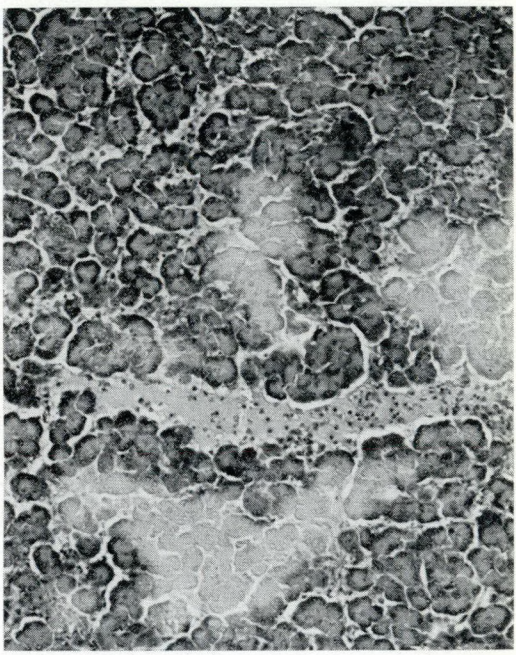

Fig. 3.—Microscopic appearance of the pancreas 20 hours after the Pfeffer procedure.

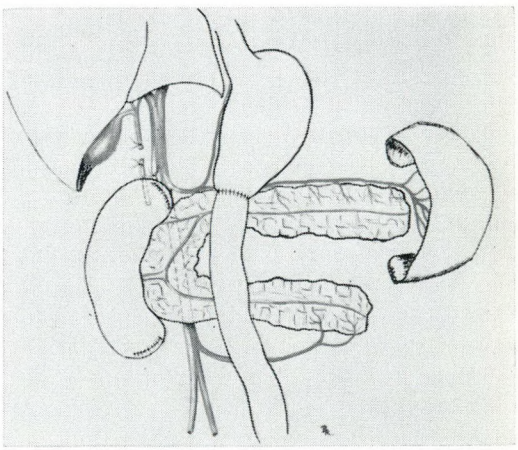

Fig. 4.—Diagram of the Pfeffer procedure with ligature of the superior pancreaticoduodenal vein.

dogs. Two of the dogs died 19 hours after operation and the other eight, all of whom were in profound shock, were killed at 20 hours. Autopsies were performed immediately after death. After taking photographs, the duodenal loop and pancreas were fixed in formalin with as little delay as possible so as to minimize post-mortem autolysis. Eight of the 10 dogs had developed total hemorrhagic pancreatitis and in the remaining two, over 80% of the gland

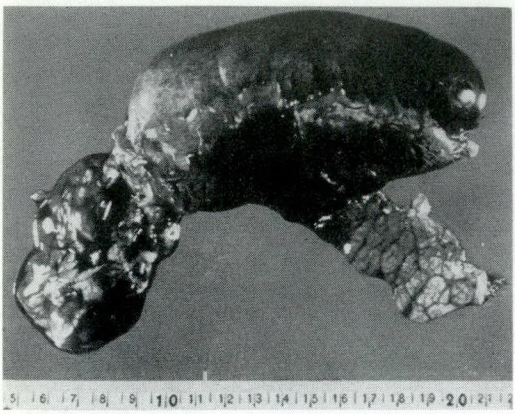

Fig. 5.—Extensive hemorrhagic pancreatitis produced by combining the Pfeffer procedure with ligature of the superior pancreaticoduodenal vein.

was affected. The pancreatitis was associated with a tense duodenum, the wall of which was thickened, red and congested, but no evidence of necrosis or incipient perforation was found (Fig. 5). The pancreas was not adherent to adjacent structures and invariably a blood-stained peritoneal exudate was present, averaging 400 ml. in volume. Cultures taken from this fluid were sterile. Half of the dogs in the group were given large doses of penicillin before and after operation, and the duodenal segment was irrigated with 0.5% neomycin solution, instead of saline, before closure. However this treatment had no influence whatsoever on the development or severity of the subsequent pancreatitis. The pressure within the distended duodenum was measured by means of an obliquely inserted needle connected to a manometer. The values ranged from 480 to 670 mm. with an average of 610 mm. of water. Microscopically, the pancreas showed a dense infiltration of the interstitial spaces with red blood cells and a few polymorphs associated with widespread coagulation necrosis of the parenchyma (Fig. 6). The high-power view emphasizes the coagulated cytoplasm and lysis of the parenchymatous nuclei (Fig. 7). These results suggest that duodenal distension combined with some degree of venous stasis in the pancreas is an efficient means of producing hemorrhagic pancreatitis. A similar mechanism might explain the occurrence of hemorrhagic pancreatitis after gastrectomy with gastrojejunal anastomosis, should afferent loop obstruction and distension develop.

Polya gastrectomy was performed upon

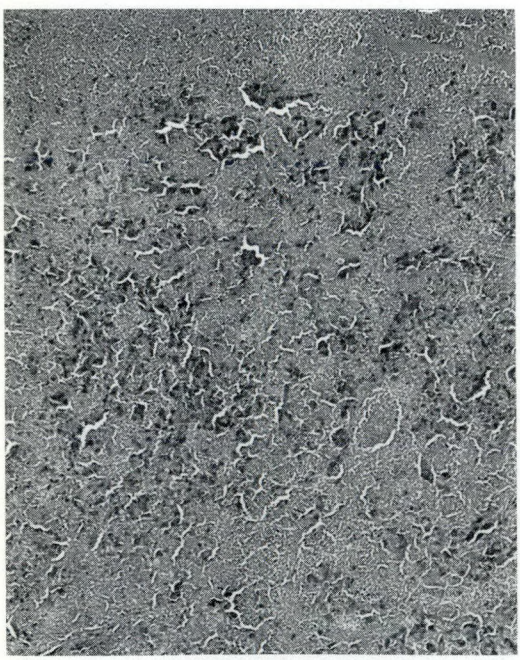

Fig. 6

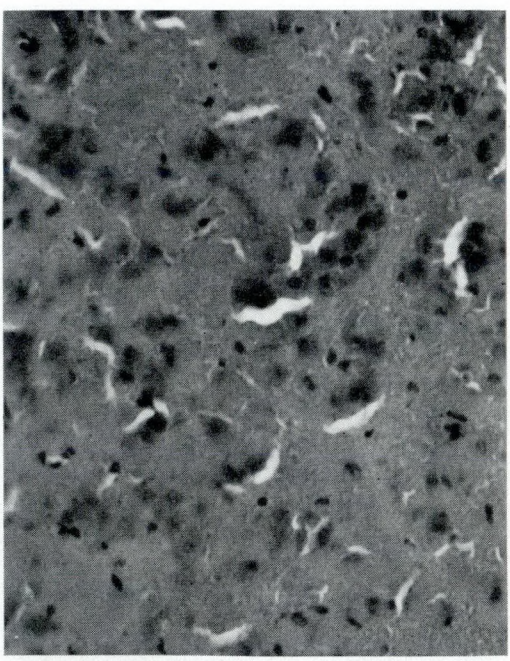

Fig. 7

Figs. 6 and 7.—Low and high-power views of the hemorrhagic pancreatitis produced by the Pfeffer procedure and ligature of the superior pancreaticoduodenal vein.

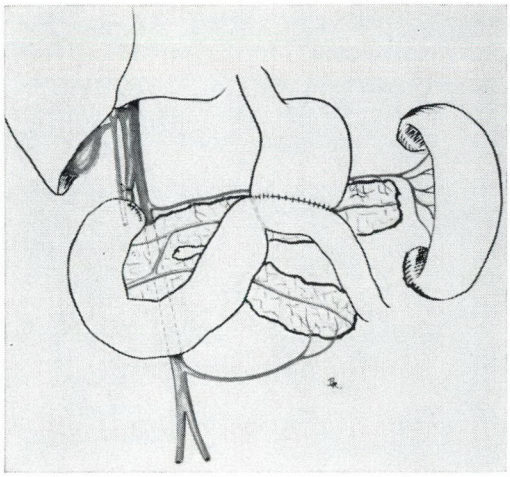

Fig. 8.—Diagram of Polya gastrectomy and ligature of the superior pancreaticoduodenal vein indicating the site of afferent loop obstruction.

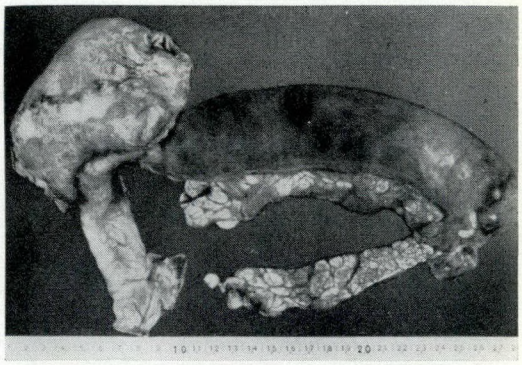

Fig. 9.—Stomach with afferent and efferent loops and pancreas 20 hours after Polya gastrectomy, vein ligation and moderate obstruction of the afferent loop. Hemorrhagic pancreatitis confined to the head of the gland.

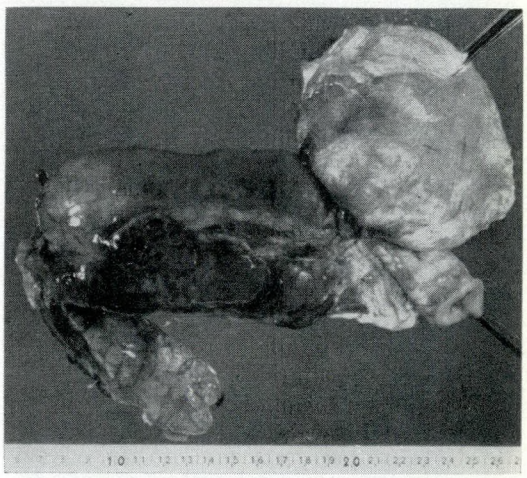

Fig. 10.—Stomach with afferent and efferent loops and pancreas 20 hours after Polya gastrectomy with severe obstruction of the afferent loop. Extensive hemorrhagic pancreatitis involving most of the gland.

10 dogs using a short afferent loop 12 cm. in length and a right-to-left anticolic gastrojejunal anastomosis. After closure of the duodenal stump, the superior pancreaticoduodenal vein was tied. In order to simulate clinical conditions, the common bile duct was not interrupted (Fig. 8). Varying degrees of obstruction were produced in the afferent loop close to the stoma using different methods. The site of the obstruction is shown by the dotted line on the diagram. Moderate obstruction was achieved by an excessive turn-in at the anastomosis and by hitching the afferent loop along the lesser curvature. Severe obstruction was obtained by inserting a submucosal purse-string suture around a piece of corrugated rubber drain within the lumen. Complete obstruction was achieved by a series of through-and-through mattress sutures protected by seromuscular sutures to avoid leakage through the stitch holes. Twenty hours after operation the dogs were killed. All had developed hemorrhagic pancreatitis, varying from disease localized to the head of the gland only (Fig. 9) to total involvement of the pancreas (Fig. 10). It was found that the extent and severity of the pancreatitis depended on the degree of duodenal distension which in turn depended on the completeness of the obstruction to the afferent loop. Microscopically, the moderately affected glands showed extensive infiltration of the interstitial spaces with an inflammatory exudate containing many polymorphs and early degenerative changes in the parenchymal cells (Fig. 11). The severely affected glands showed extensive interstitial hemorrhage with coagulation necrosis and many areas presented as an acidophilic amorphous mass with a few basophilic nuclear remnants (Fig. 12).

Under these experimental conditions, three possible factors contribute to the etiology of the pancreatitis — venous stasis, duodenal distension and possible reflux of duodenal contents into the pancreatic duct system. In order to assess their relative im-

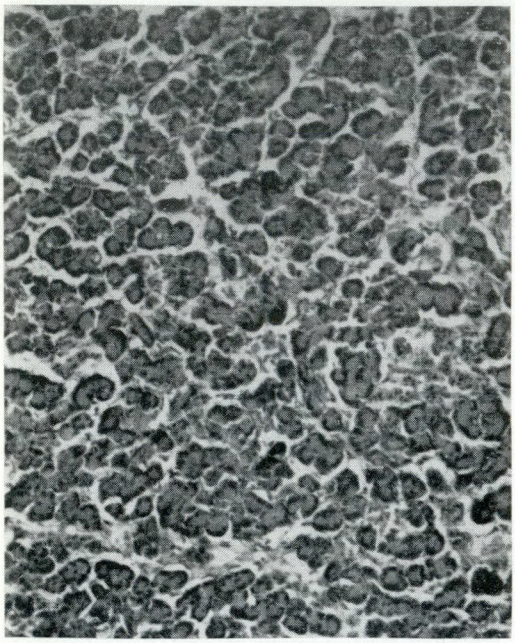

Fig. 11.—Microscopic appearance of the pancreas shown in Fig. 9.

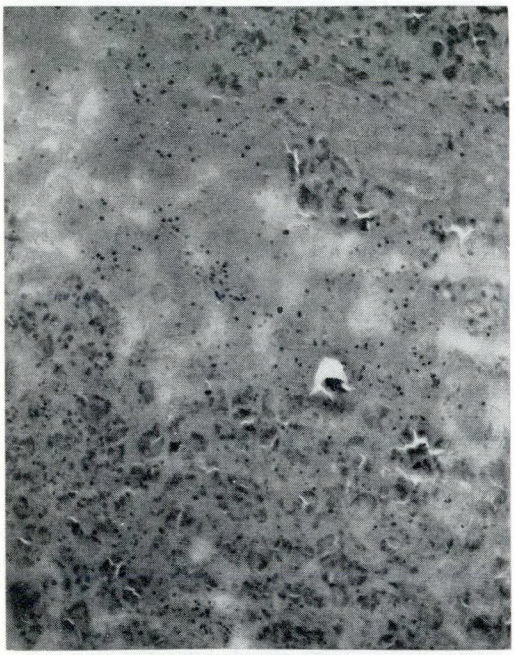

Fig. 12.—Microscopic appearance of the pancreas shown in Fig. 10.

portance, further experiments were carried out.

Polya gastrectomy was performed on five dogs with ligature of the superior pancreaticoduodenal vein but without any obstruction to the afferent loop (Fig. 13). All of the dogs remained in a satisfactory general condition after the operation and at 20 hours they were killed. Autopsy revealed some edema and congestion of the head of the pancreas but the lobular structure of the organ was preserved (Fig. 14). The edema was obvious on microscopical examination but there was no cellular infiltration and no sign of any damage to the parenchymal cells, which appeared healthy with normal vesicular nuclei and unaltered cytoplasm. Hence, venous stasis alone is not a dominant factor.

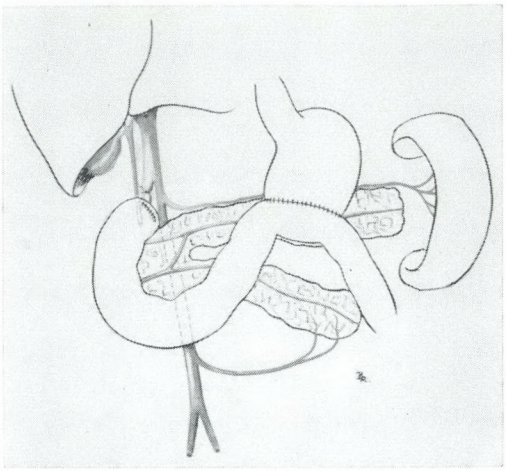

Fig. 13.—Diagram of Polya gastrectomy with ligature of the superior pancreaticoduodenal vein.

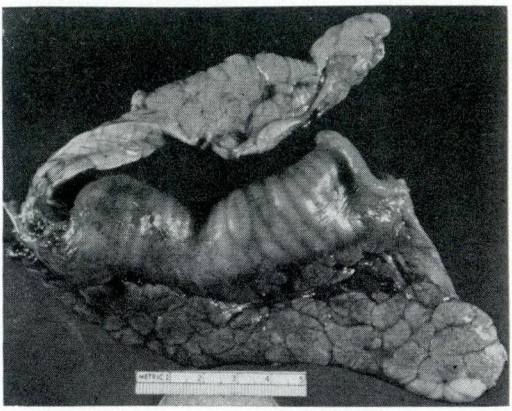

Fig. 14.—Edema and congestion in the head of the pancreas produced by conditions illustrated in Fig. 13.

An isolated duodenal loop was prepared in another group of five dogs. The common bile duct was divided, the superior pancreaticoduodenal vein tied and both the

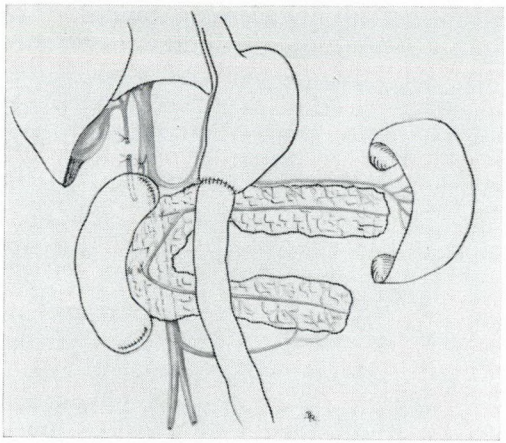

Fig. 15.—Diagram of the Pfeffer procedure combined with ligature of the superior pancreaticoduodenal vein and both pancreatic ducts.

pancreatic ducts divided (Fig. 15). Thirty-six hours later the dogs were killed. Autopsy showed that none of the dogs had developed hemorrhagic pancreatitis. Instead, the glands were swollen and extremely friable, and extensive fat necrosis was present over and around the pancreas. In addition, a remarkable adhesion of the surrounding organs to the pancreas was noted. Another interesting feature was the absence of any significant peritoneal exudate. Histological examination showed extensive edema of the pancreas with a meagre infiltration of polymorphs and early degenerative changes in the parenchymal cells (Fig. 16). Typical areas of fat necrosis were seen on the surface of the pancreas. A similar procedure was carried out in five more dogs but increase in pressure within the pancreatic ducts was prevented by inserting a fine polyethylene cannula into the duct and allowing free drainage (Fig. 17). Under these conditions no important change occurred in the gland except for some edema and congestion around the head of the pancreas.

Therefore, it would appear that venous stasis in the pancreas combined with distension of the duodenum are in themselves insufficient to cause acute hemorrhagic pancreatitis under these experimental conditions. There must also be reflux of duodenal content into the pancreatic duct system. Possibly this reflux results in the activation of proteolytic enzymes in the pancreatic juice leading to diffuse interstitial hemorrhage and rapid coagulation necrosis. Alternatively, the effect of the reflux may simply be due to the transfer of high intraduodenal pressure into the pancreatic ducts.

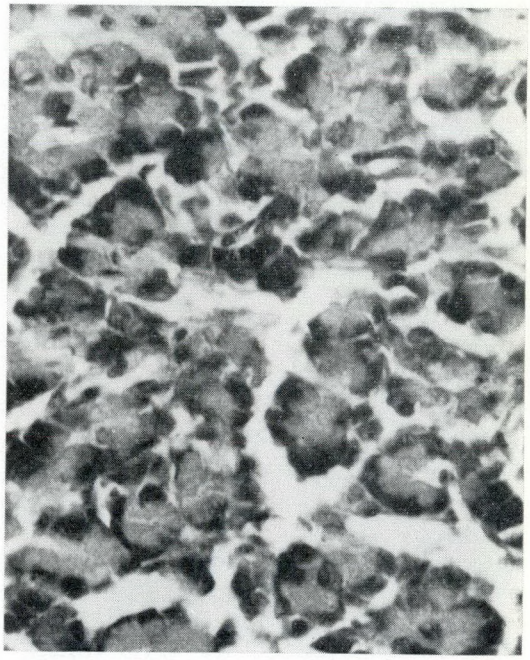

Fig. 16.—Microscopic appearance of changes in the pancreas produced by conditions illustrated in Fig. 15.

All of our experiments were carried out on mongrel dogs varying from 7.5 to 12 kg.

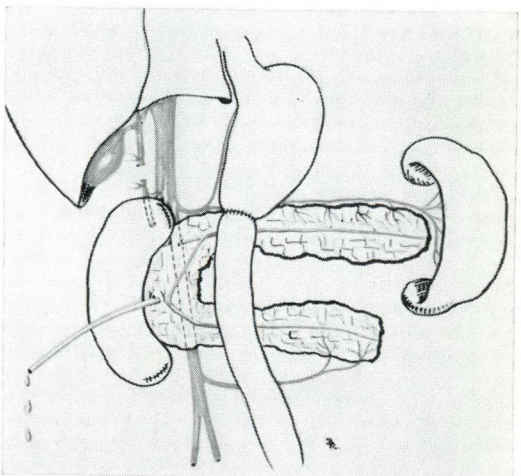

Fig. 17.—Diagram of the Pfeffer procedure combined with ligature of the superior pancreaticoduodenal vein and both pancreatic ducts with decompression of the pancreas with a fine polyethylene tube.

in weight. They were given water but no food for 24 hours before operation and nothing by mouth after operation. During the operative procedure they received 500 ml. of 5% dextrose in saline and a further 500 ml. of the same solution in the first postoperative day.

Discussion

About half of the cases of pancreatitis that occur after gastrectomy develop in the first four postoperative days. This is a time when gastrointestinal secretions are depressed and there has been little or no stimulation from ingested food. Millbourn[4] summarized the possible etiological mechanisms in the following five points: 1. Mechanical injury to pancreatic tissue during operation owing to traction or pressure sufficient to cause laceration. 2. Vascular damage with resulting ischemia in the gland. 3. Stagnation in the duodenum and reflux of duodenal contents or of bile into the pancreatic ducts. 4. Spasm of the pancreatic sphincters. 5. Injury to a pancreatic duct.

Warren[10] has pointed out that damage to the pancreatic ducts or pancreas at operation does not necessarily lead to pancreatitis, while severe pancreatitis may follow gastrectomy with little or no disturbance to the gland. The experimental work described in this paper suggests that duodenal distension with reflux of duodenal content into the pancreas associated with some venous stasis in the gland will lead to acute hemorrhagic pancreatitis. Perman,[7] in 1935, as a result of clinical observations, had suggested this hypothesis. Recently, McCutcheon and Race[3] were able to demonstrate very nicely that pancreatitis following Pfeffer's procedure was dependent upon free communication between the tense distended duodenal loop and the pancreatic ducts. They injected barium sulfate suspension into the duodenum and were able to recognize barium deposits in the substance of the diseased pancreas.

Anderson and Bergan[1] were able to show that trypsin and whole blood, when incubated for 24 hours, resulted in the production of a toxic substance which will produce acute hemorrhagic pancreatitis when injected into the pancreatic duct. In spite of this, we have no definite evidence that the reflux of duodenal content into the pancreas in our experiments produces changes by activating interstitial enzyme digestion. It may be purely a pressure effect within the pancreas.

It is likely that many leakages from duodenal stumps after Polya gastrectomy, and perhaps all such complications, are preceded by distension of the duodenum. This may be secondary to obstruction of the afferent loop close to the stoma and Leslie[2] has evidence that functional obstruction can occur just beyond the duodenojejunal flexure in the absence of any obvious mechanical factor. If the duodenal stump closure had been satisfactory with a good blood supply, it is possible that the suture line would withstand high pressures for several hours, and if, at the same time, there had been some interference with the venous return from the pancreas as a result of a long-standing ulcer or its removal, a condition closely resembling our experimental operation would then exist.

Such a development could only occur after gastrectomy with gastrojejunal anastomosis. Wallensten[9] found that acute postoperative pancreatitis resulted in 12 deaths in a series of 1769 Billroth II resections, while there were no deaths caused by acute postoperative pancreatitis in 605 Billroth I resections. Pendower and Tanner[6] described nine cases of postoperative pancreatitis after gastrectomy and stated that collected figures from various authors show that post-gastrectomy pancreatitis has a mortality of over 50%. All of these authors emphasize the fact that post-gastrectomy pancreatitis occurs only after the Polya type of operation.

Summary

A series of experimental operations on dogs are described which suggest that venous congestion in the pancreas, distension of the duodenum and reflux of duodenal content into the pancreatic ducts play an important role in the etiology of acute hemorrhagic pancreatitis complicating Polya gastrectomy. The possible clinical significance of these findings is discussed.

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Résumé

La pathogénie de la pancréatite hémorragique aiguë est toujours peu comprise. Les auteurs présentent ici une technique expérimentale, adaptable au chien, permettant de reproduire l'affection. Leur méthode dérive de celle qui fut décrite par Pfeffer; un segment de duodénum de 7 à 10 cm. de longueur et comprenant l'abouchement des canaux pancréatiques est isolé et fermé à son extrémité; de plus le canal cholédoque est disséqué et l'extrémité pylorique de l'estomac est anastomosée à la partie distale du duodénum. Enfin on ligature la veine pancréatico-duodénale supérieure de façon à créer un certain trouble dans la circulation veineuse. Dans ces conditions, il se fait rapidement une dilatation de la portion suturée de duodénum et sur dix animaux d'expérience, huit furent atteints d'une pancréatite hémorragique grave. Dans une autre série d'investigations, on effectua des résections gastriques selon Polya sur des chiens; cette résection était associée à une ligature de la veine pancréatico-duodénale supérieure ou à diverses obstructions sur la partie afférente de l'intestin. Les auteurs sont amenés à conclure qu'une stase veineuse intra-pancréatique associée à une dilatation du duodénum est insuffisante pour créer une pancréatite. Il faut encore que le contenu duodénal reflue dans le canal pancréatique. Les relations de ces expériences avec les constatations cliniques après gastrectomie selon Polya sont discutées.

THE BIOLOGY AND TREATMENT OF INTRACRANIAL TUMORS. 9th Annual Scientific Meeting of The Houston Neurological Society. Compiled and edited by William S. Fields and Paul C. Sharkey. 505 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1962. $17.25.

The editors, Drs. Fields and Sharkey, have assembled a group of experts to write on various aspects of the study and treatment of brain tumours. The result is a collection of chapters, each of which is followed by the short discussions that took place when the papers were originally presented at the Ninth Annual Scientific Meeting of The Houston Neurological Society. The participants, most of whom are well recognized, write about current basic research, pathology, diagnostic techniques and treatment of brain tumours. The book is not comprehensive, but describes many important facets of pathology and clinical management. Recent experimental work is covered in detail. The chapters are unevenly balanced and there is little continuity between them. For example there is one chapter on angiography in brain tumours and one on the activity of meningioma whorls *in vitro*. This disadvantage, inherent in the symposium-style volume, is offset by the excellence of the majority of the individual contributions.

The classification of brain tumours based on light-microscopy studies and clinical follow-ups is discussed by K. J. Zulch. This is complemented by a description of the electron microscopy of these tumours by S. A. Luce. Dr. Zulch explains the problems in classification and nomenclature and indicates changes that may be accepted when more knowledge is acquired. Two other chapters that deserve special mention are those on the surgical management of glial tumours by C. S. MacCarty, and the radiation therapy of intracranial neoplasms by J. Bouchard. Both of these are concerned with the long-term results of treatment and are based on series of patients treated in a uniform manner.

Neurosurgeons and neuropathologists will find the book stimulating and useful.

MECHANICAL INVESTIGATION OF EXPERIMENTAL FRACTURES*

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Montreal

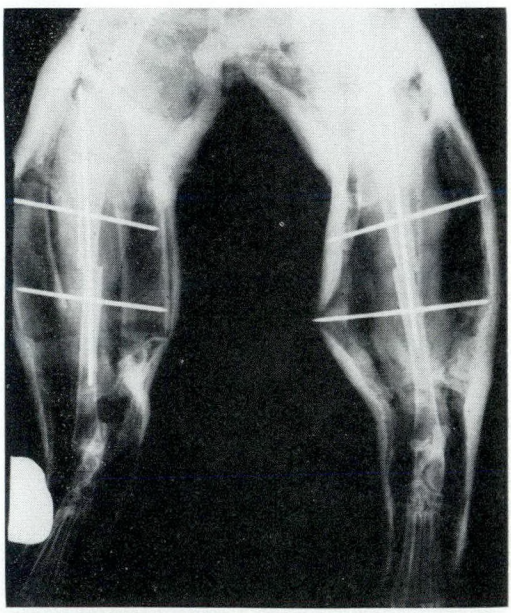
THESE animal experiments were undertaken to clarify two specific aspects of fracture healing in tubular bones. The first objective was to determine the rate of normal fracture healing in tubular bones; in other words, does a fresh fracture consolidate at a constant rate, or is there a well-defined phase of strengthening during which the fracture line rapidly becomes stable, much like the sudden formation of a crystal from its liquid state? The second objective was to analyze the effect of compression on the healing process of fresh fractures in tubular bones.[3] Almost as a corollary, the experiment was later modified to investigate (a) the precise function of the periosteum in fracture healing; (b) the origin of the so-called periosteal callus.

PROCEDURE

The experiment was performed on rabbits with midshaft tibial fractures. Numerous precautions were taken to standardize the fracture lines and to treat them identically. To achieve this control, the tibias were transversely transected at a constant midshaft level, using an electric saw; the fracture line was immobilized using an intramedullary Kirschner pin, as well as two transverse pins, which were introduced at a prefixed distance, on either side of the fracture line. The pins were incorporated in the plaster spica to provide a sound well-standardized immobilization (Fig. 1a). The reduction was controlled radiologically (Fig. 1b) at regular intervals, and fractures which were not identical in type or treatment were later excluded.

The stability of the healing fracture line was tested in the following manner. The animals were sacrificed, the plaster spica was removed and the intramedullary pin was carefully extracted. The proximal tibial fragment was firmly fixed in a vice, and

a



b

Fig. 1.—Method of immobilization: (a) Bilateral hip spica incorporating the transverse pins in the plaster. Note the polythene window on the right tibia. (b) Control radiograph. Note that the pins on the right tibia are not incorporated in the plaster to allow compression. This animal belongs to the compression experiment.

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a container was suspended from the distal transverse pin (Fig. 2); the distance between the transverse pins and the fracture line was constant. Sand was then slowly poured in the container until the callus broke. The weight of sand was recorded and was considered to represent the strength of the callus at that precise moment. By sacrificing the animals at different intervals, it was possible to study the gradual strengthening of the fracture line.

One might question whether a transverse shearing force is a fair test of the callus strength or, indeed, whether a mechanical evaluation of a callus is a sound method of assessing its maturity. This method of evaluating the callus strength admittedly does not reproduce the precise clinical stresses sustained by a healing fracture; nevertheless, the experiment permitted a comparison between calluses of increasing maturity by providing a mechanical and numerical evaluation of each fracture line.

By recording the age of the callus horizontally and the strength of the callus vertically, it was possible to record every fracture graphically. The different markings on the graph (Fig. 3) represent 194 such experimental fractures. The resultant curve represents the rate of healing in a fresh tibial fracture. The curve is readily divided into three sections, each representing approximately three weeks' duration. The first section is mostly horizontal, the second is almost vertical, before the curve finally levels off at the end. After 60 days, the fracture is clinically united. Indeed more prolonged immobilization does not yield a much stronger callus. In fact, 50 lb. suspended from a similarly located transverse pin will break the intact tibia of a rabbit. This small control experiment was repeated on 30 intact tibias; hence, with these terms of reference, 50 lb. represents the strength of an intact tibia. The graph (Fig. 3) illustrates that such a stability or strength is already present in a healing fracture at 60 days.

If one considers the fracture line mechanically, the graph may be divided into three periods of increasing solidity: first, a period of instability; later a period of plasticity, and then a period of rigidity (Fig. 4). These three periods were correlated with the gross and microscopical analyses of the callus. The three periods, which precede complete bony union, have a close time relationship with very definite phases of fracture healing (Fig. 5). First, there is the fracture hematoma period or phase of pre-callus, which corresponds to the period of instability; then, there is the period of periosteal callus formation, during which the fracture line rapidly gains strength; during the third phase, endosteal callus formation is observed, adding surprisingly little stability to the fracture line. At this point, 60 days after the beginning of the experiment, the fracture is clinically

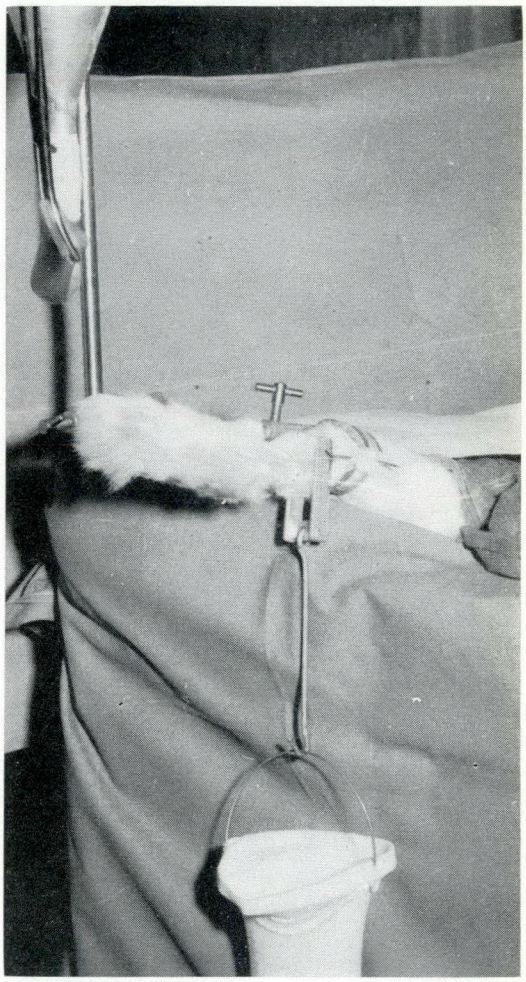


Fig. 2.—The "re-fracture" experiment. The proximal fragment is fixed in a vice, and a container is suspended from the distal transverse pin. The container is then gradually filled with sand via the funnel until the callus is "re-fractured".

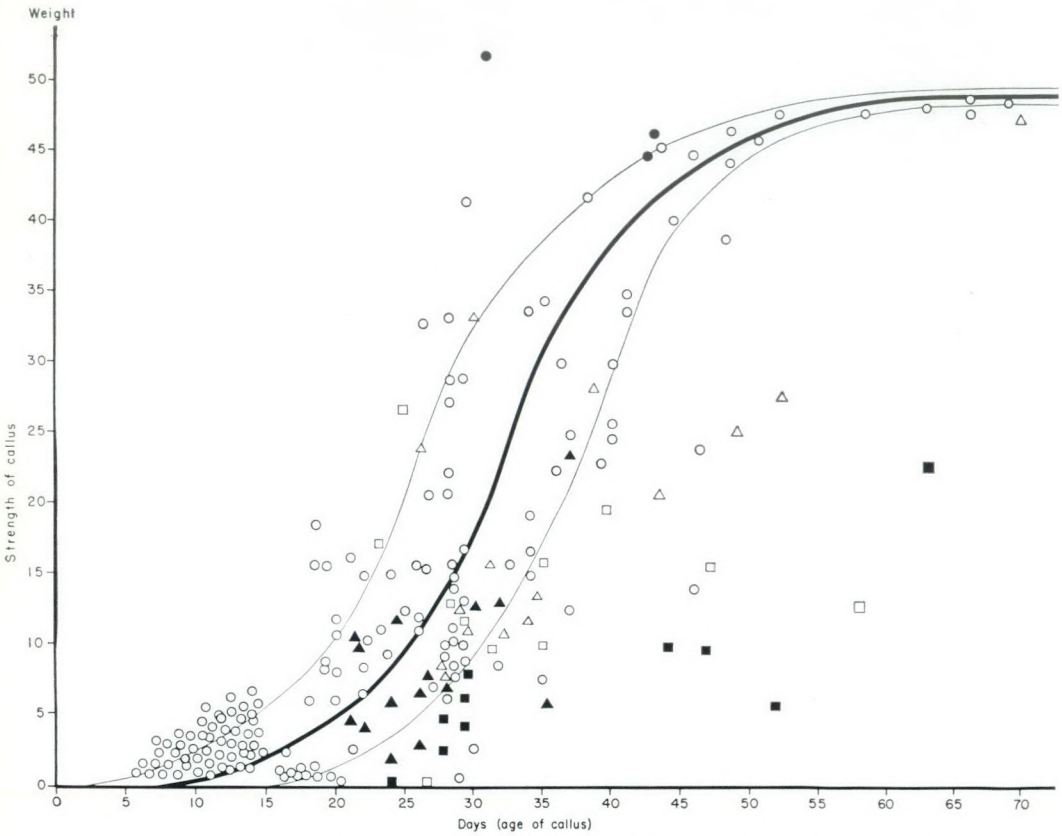


Fig. 3.—Graph of 194 experimental fractures demonstrating the *rate* of increasing stability in a maturing fracture callus. The white circles represent the strength of comparable transverse midshaft fractures at different stages of fracture healing; the black squares represent animals suffering from pasteurellosis, in whom the infection extended to the fracture line and delayed the consolidation. As would be expected, these fractures do not fall within the curve of normal fracture healing, but well below it. The black circles represent comminuted but stable fractures, while the white squares represent unstable comminuted fractures. Both fractures had a predictable influence on fracture healing; the stable comminuted fractures hastened the healing process, while the unstable comminuted fractures delayed the healing.

The fractures designated by the black and the white triangles indicate fractures which were treated by compression and represent an experiment that will be discussed later. Note that fractures treated by compression were not stronger than control fractures of the same age. The white triangles represent tibias which were compressed two weeks after they had been fractured; the black triangles represent experiments where the compression was applied from the very beginning.

solid, and the cortex is sandwiched between an outside, sometimes exuberant periosteal callus, and a more discrete endosteal callus. The early clinical union is entirely dependent on this callus "sandwich", since the underlying cortex is dead on either side of the fracture line. Indeed, unless the bone is cut longitudinally, the necrotic bone ends may be entirely concealed by the periosteal callus (Figs. 6a, c and Figs. 9a, b).

At 10 days, one observes the beginning of the periosteal callus (Fig. 7). The point of origin is most constant; it is never adjacent to the fracture line, but 1 cm. or so away from it. The callus usually assumes a triangular shape, with its widest measurement at the level of the fracture line; the vector of growth is outwards and towards its mate on the opposite fragment. These two triangular calluses are first joined by fibrocartilaginous tissue which is less mature than the callus proper, with a line of junction usually located at the level of the fracture line. By the fourth week, the bridge is usually complete, although the callus is still mostly fibrous; during the plasticity phase, within the first three or

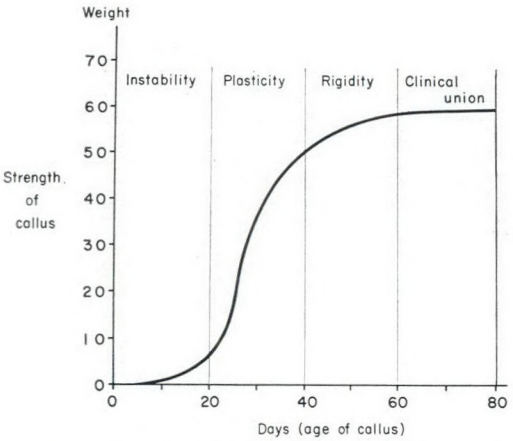


Fig. 4.—Graph of three phases of solidity. The "healing curve" of a fresh fracture callus may be divided into three stages of increasing stability; each period lasts approximately three weeks. (See text for details.)

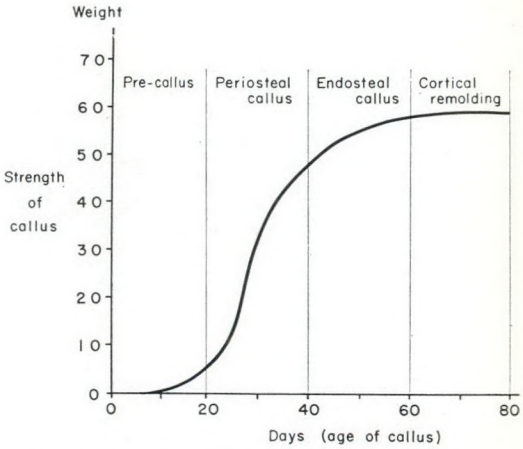


Fig. 5.—Graph of three phases of callus formation. The "healing curve" of a fresh fracture may be divided into different stages of callus formation.

four weeks, the callus attains its maximal mature size. The next phase is one of calcification within this large fibrous callus. The process of calcification is multicentric and proceeds in an apparently irregular fashion throughout the whole callus. The final process is one of ossification. A similar chain reaction begins in the endosteum, approximately five weeks after the fracture.

Compression of Fresh Experimental Fractures

Having analyzed the manner and rate of normal fracture healing in rabbits, the experiment was then modified to analyze the effect of compression on the callus formation of fresh tubular bone fractures.

The influence of compression was experimentally investigated in the following manner. The control tibia was first fractured and immobilized as already described; the fracture line of the opposite tibia was compressed by elastic bands applied to the transverse pins (Figs. 8a, b); the transverse pins on the "compression side" were covered by a polythene tube and were thus not incorporated in the plaster spica. Hence, it was possible to compare elastic compression on one side versus rigid complete immobilization on the other.

This experiment was performed on 30 rabbits. The force of the compression and the timing of application of the compression were controlled and systematically altered. Whether the compression was applied at the time of the experimental fracture or two weeks later, or whether the compression force was great or small, the authors could not demonstrate any beneficial influence of compression on fracture healing.

The graph (Fig. 3) shows that compression, immediate or delayed, does not hasten the consolidation of fresh tubular bone fractures. Charnley[1] had suggested that this was so and these experiments confirm it. Compression is undoubtedly useful for the apposition of spongy bone surfaces, where consolidation takes place by endosteal callus formation at the points of contact.[2] Under these circumstances, compression not only improves the contact, it appears to stimulate new bone formation in accordance with Wolff's law.[5] An almost similar situation exists in cases of delayed union of tubular bones where compression can be applied to a now porous and potentially osteoblastic cortex; on some of these occasions, there is also compression of two exuberant periosteal calluses arising from each fragment. However, this is far from the state of affairs in a fresh fracture of a tubular bone; in this instance, the cortical ends are dead and inert; such a fracture first heals by a periosteal callus which by-passes the dead cortex (Fig. 9). Com-

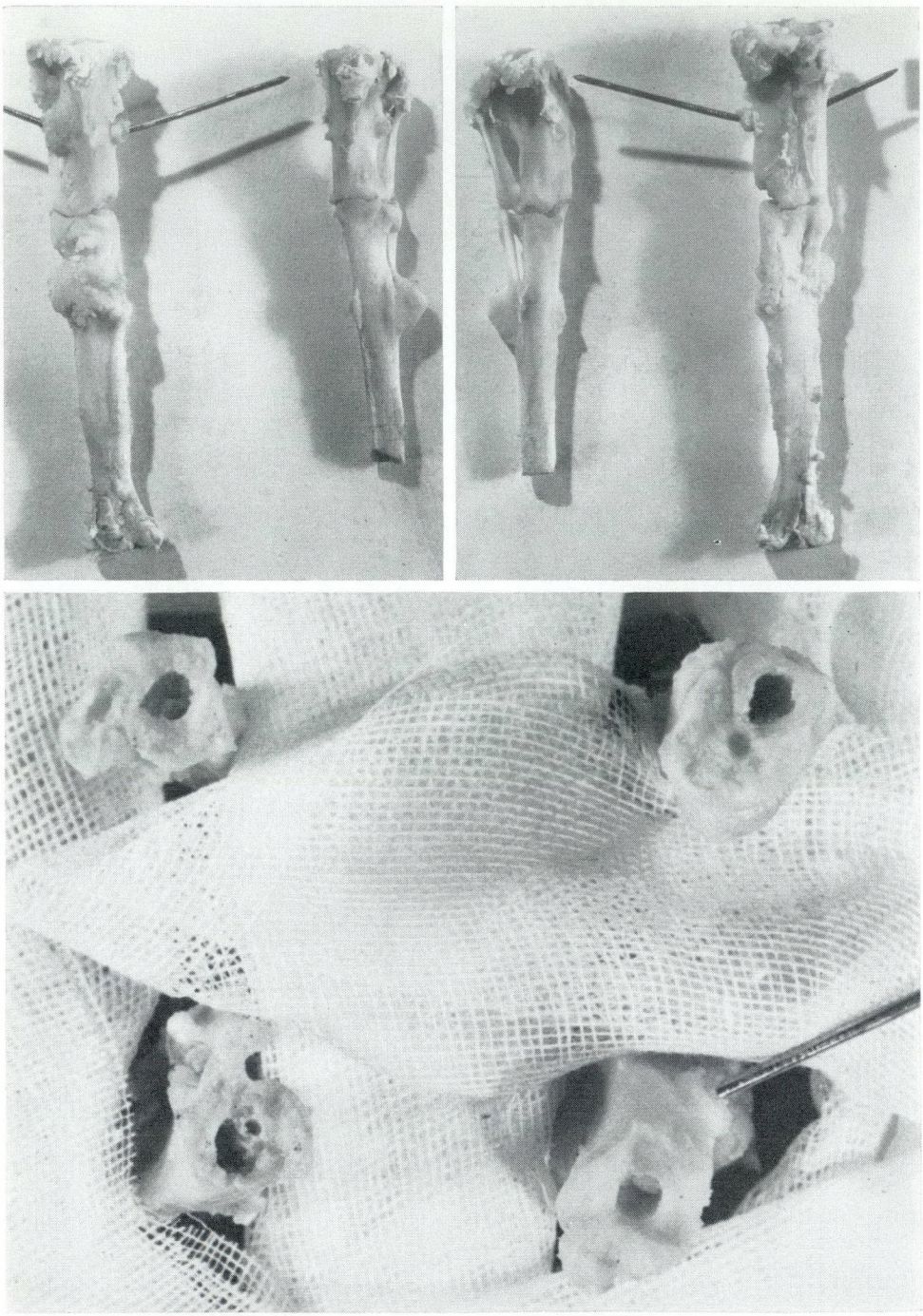


Fig. 6.—Callus by-pass (specimen No. 90). Experimental callus after 66 days (refractured with 55 lb., i.e. "normal" strength): (a) anterior; (b) posterior; (c) cross-sectional.

Note: 1. Outer callus is more abundant on lateral and posterior surfaces of tibia in spite of circumferential and extraperiosteal dissections. 2. Inner callus is present around the intramedullary pin on one side only (Fig. 6c); there is almost no inner callus inside opposite fragment where the longitudinal pin was not within the medullary cavity. 3. Inert cortex (more obvious in anteroposterior view of specimen with transverse pin still *in situ* (Fig. 6a)) partially concealed by outer callus "by-pass". 4. The "pin-reaction" around the transverse pins (Fig. 6a, b): these pins were particularly loose and stimulated an exuberant "pin-reaction"; this is consistently more abundant on the peroneal side of the tibia (compare with Fig. 10).

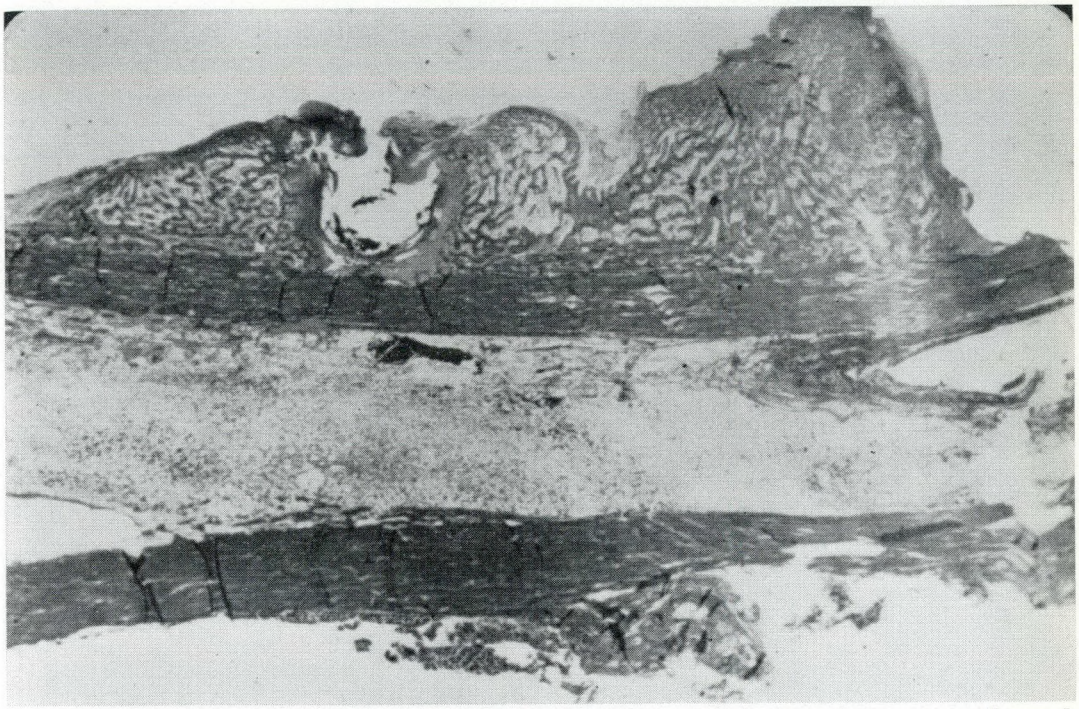
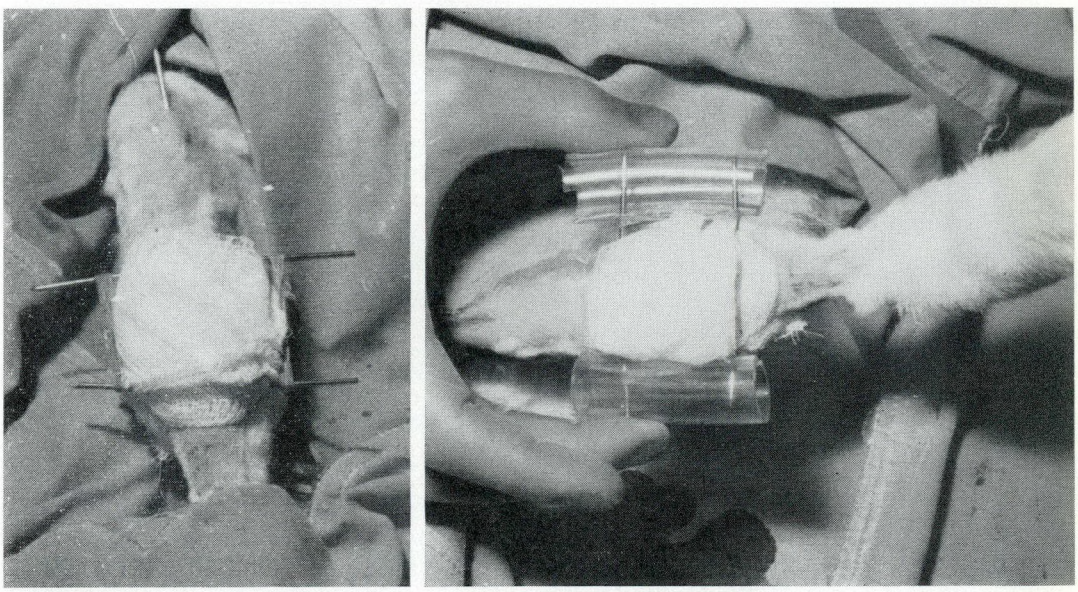


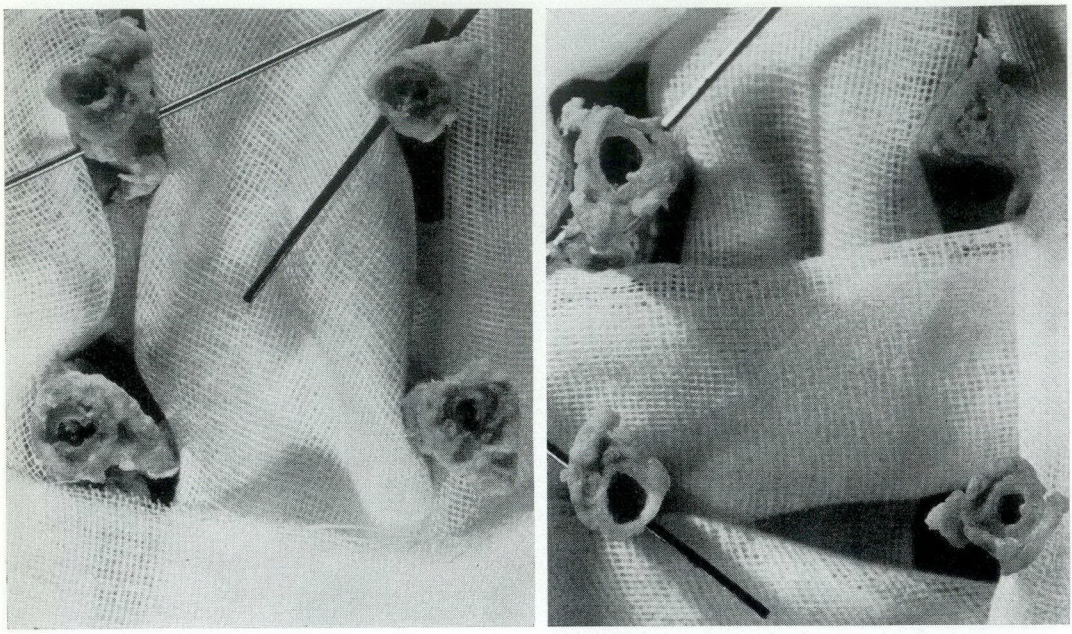
Fig. 7.—Shape of outer callus after 20 days. Note the point of origin of the outer callus and the absence of inner callus at this stage.



a b

Fig. 8.—Elastic compression of the fracture: (a) elastic bands in place. (b) Polythene cover to isolate the transverse pins from the plaster. Note the "polythene window" (Fig. 1a) which allows the observation of the compression bands during the experiment.

pression of dead cortical surfaces in fresh tubular fractures does not favour the development of such a by-pass;[4] on the other hand, it should not interfere with the formation of this periosteal callus. Hence, a compressed fresh fracture of a tubular bone heals at a normal rate, as clearly shown in the graph.



b a

Fig. 9.—Outer callus by-pass: cross-sectional views. The calluses are (a) 20 and (b) 30 days old. Note: 1. Outer callus by-pass (thicker posteriorly and laterally). 2. Sluggish inner callus. 3. Inert cortical surfaces.

Comment

It has already been noted, in experimental fractures of the rabbit at least, that the periosteal callus contributes most of the early stability in a healing fracture. Certain observations made during this experiment have led the authors to speculate on the precise origin of this outer callus. Three such observations will be briefly discussed: first, the location of the periosteal callus; second, the new bone formation noted around the transverse pins, what has been called the "pin reaction"; and third, the shape of the outer callus, noted in the presence of bayonet apposition of a fracture line.

1. *The Location of the Periosteal Callus*

The periosteal callus was consistently more abundant on the posterior and posterolateral surfaces of the tibia; it was almost never noted anteromedially (Fig. 10 and Figs. 6 and 9). This phenomenon was originally attributed to stripping of the periosteum at the time of the experimental fracture; however, the observations were identical even when the dissection was entirely extraperiosteal. It would therefore appear that the absence of the outer callus on the anteromedial side of the tibia has little to do with the stripping of the periosteum. Rather, the location of this outer callus seems to depend on a tissue which is outside the periosteal sheath and which is more abundant or more active on the posterior and lateral surface of the tibia.

2. *Pin Reaction*

The above interpretation is supported by the "pin reaction" which was consistently more abundant on the peroneal side of the tibia (Figs. 6a, 10). A tentative explanation of this phenomenon suggested that the pins elevated the periosteum on the fibular side of the tibia following insertion of the transverse pins from the tibial side. However, insertion of the same pins from fibular side yielded the same results. This unequal "pin reaction" is now better explained on a basis of greater vascularity and superior osteoblastic activity of the parosteal tissues* on the

*The parosteal tissue refers to the undifferentiated, potentially osteoblastic tissue which surrounds a tubular bone; this tissue includes the periosteum as well as the connective tissue beyond it.

fibular side of the tibia. Again, there appears to be a greater osteoblastic potential where bone is not superficial but where it is covered by muscle.

Indeed, the "pin reaction" behaves very much like the callus bridge of a healing fracture. Two conditions are essential for its development: first, a sensitive vascular parosteal tissue and, secondly, a stimulus or, in this case, movement. For example, transverse pins inserted across an intact tibia or across an intact femur hardly stimulate any "pin reaction" because in the absence of a fracture there was little if any movement. Similarly, a "pin reaction" noted above and below a fracture showed a close correlation with movement (compare Figs. 6a and 10). A tight transverse pin was never associated with a massive "pin reaction"; a loose pin, however, consistently stimulated an exuberant "callus-like" pin reaction, with a cartilaginous centre (Fig 11b). And yet, this same loose pin never initiated an equal "pin reaction" on both sides of the tibia. Hence, the two factors which influence the formation of a "pin reaction" or of an outer callus are the distribution of osteoblastic parosteal tissue and movement.

The stimulating influence of movement was also indirectly illustrated around the intramedullary pin. It has already been noted that endosteal bone formation is late to appear and that it is very sluggish in rabbits. In these experiments, new bone formation within the intramedullary canal does not begin in contact with the endosteum but around the intramedullary pin (Fig. 11a). Similarly, the transverse pins stimulated comparable transverse canals of bone around them (Fig. 11b). It would appear that new bone is formed in response to the micro movements of these pins within a potentially osteoblastic milieu.

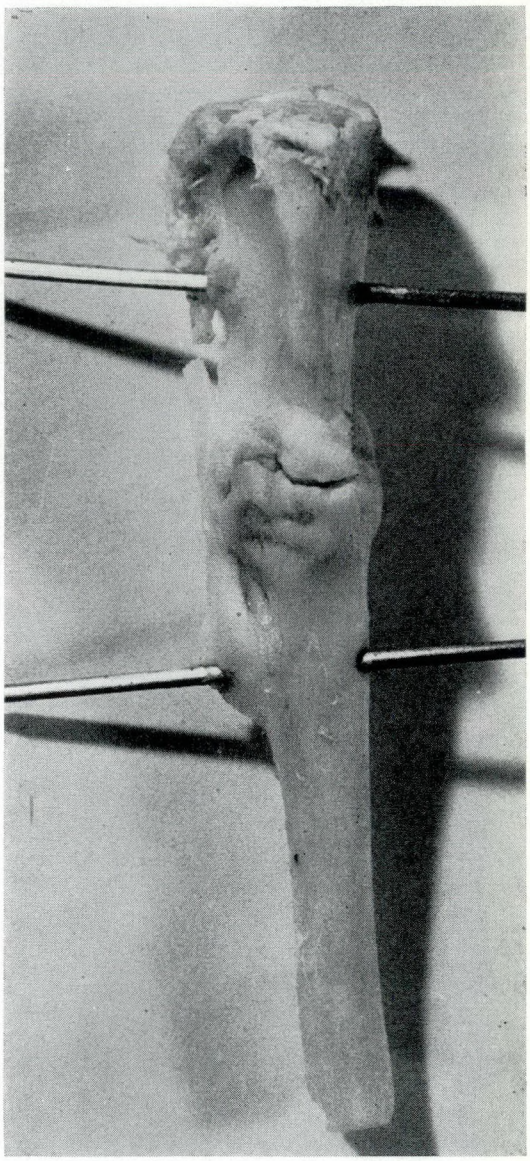
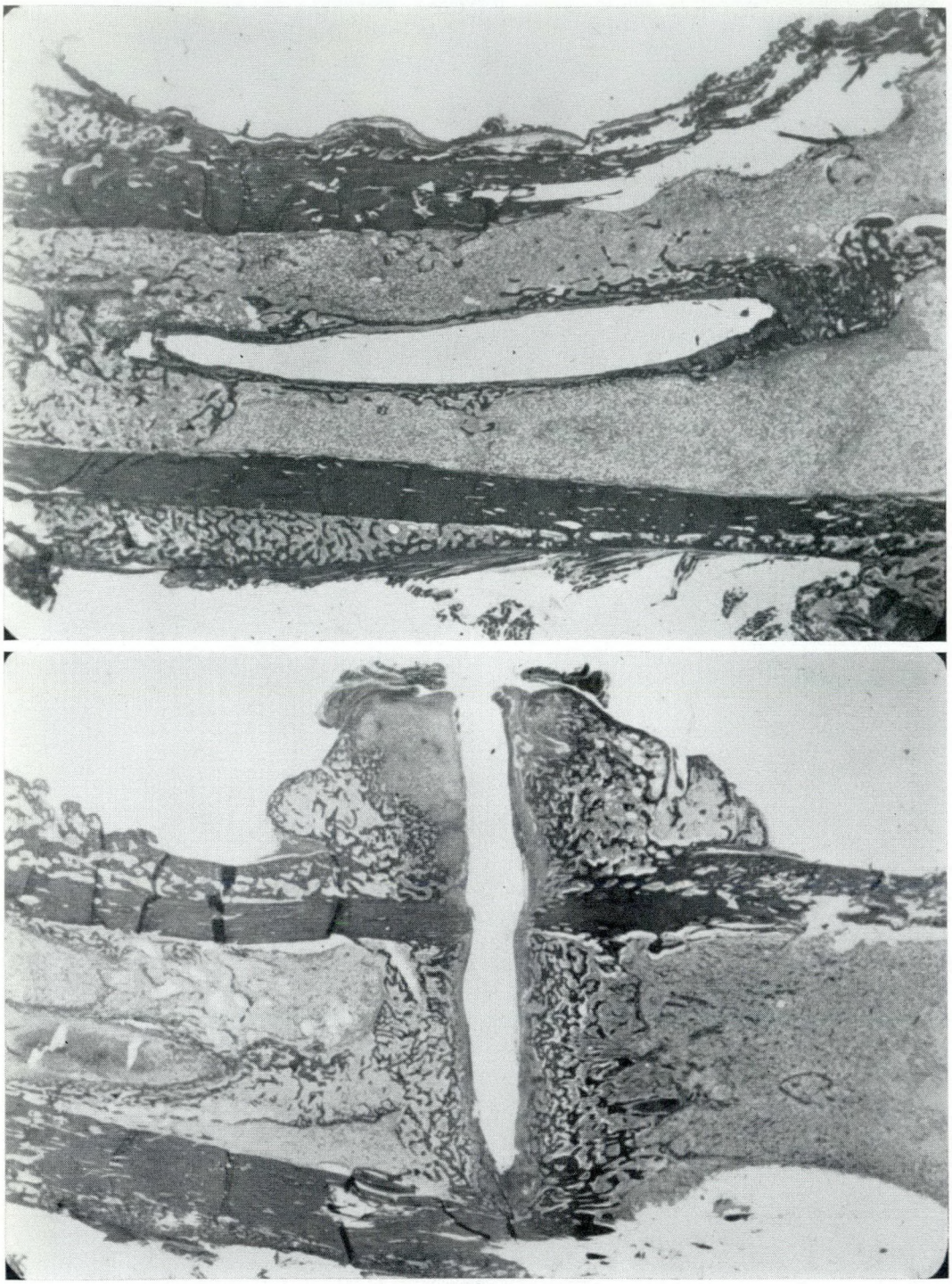


Fig. 10.—Fracture callus after 44 days. Note: 1. Outer callus is more abundant laterally and posteriorly (extra-periosteal dissection). 2. The pin-reaction is relatively limited and the transverse pins are still tight; (compare with Figs. 6a and b, where the transverse pins were loose). Note also that the pin-reaction is more abundant on the fibular side of the tibia in spite of insertion of upper pin from lateral to medial.

3. *Outer Callus and Bayonet Apposition*

In cases of bayonet apposition of a fracture line, the callus always tends to be fusiform (Fig. 12a). To achieve such a cylindrical form, one periosteal surface will be almost inert, while the periosteum and the parosteal tissue of the opposite fragment contributes most of the outer callus. A popular explanation for this phenomenon, namely, the unequal periosteal callus in the presence of the bayonet apposition, has been that subperiosteal bone formation occurs in those areas where the periosteum has been elevated from the cortex at the time of the fracture (Fig. 12b). Yet experimentally a similar callus is formed when

a



b

Fig. 11.—Endosteal callus: (a) the endosteal callus originates around the intramedullary pin and not from the endosteum. (b) Similar "bony canal" forms around the transverse pins. Observe the cartilaginous centre of the pin-reaction.

the dissection is entirely extraperiosteal. In other words, the periosteum is never elevated or separated from the underlying cortex; it is transected with the bone.

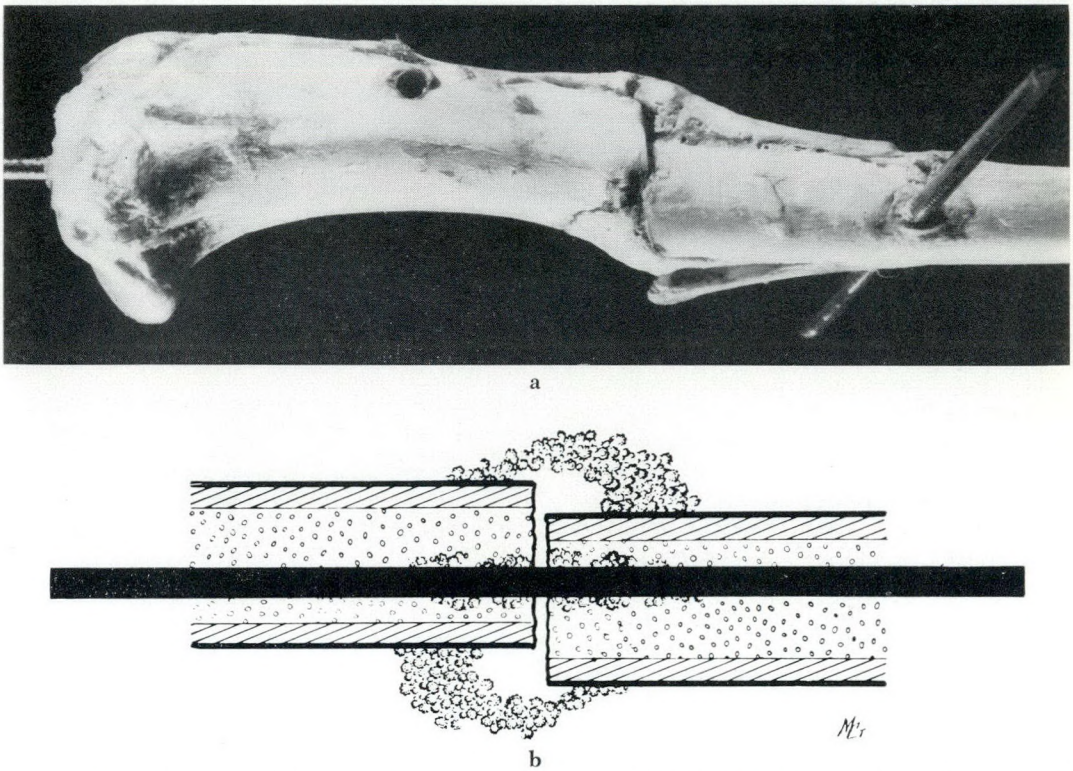


Fig. 12.—Outer callus and bayonet apposition: (a) The fusiform shape of the callus is noted in spite of extraperiosteal dissection; to achieve this fusiform shape, one periosteal surface contributes most of the outer callus while the opposite periosteal surface of the same fragment is almost inert. (b) Diagram of bayonet apposition and fusiform callus. The callus is usually presumed to develop where the periosteum has been elevated; yet experimentally (and sometimes clinically) the periosteum is known to be adherent to the cortex. (See text for details.)

Again it would appear that periosteal callus does not arise solely from osteoblastic cells situated between the cortex and the periosteum but more likely from the whole parosteal layer. Nor is periosteal stripping necessary to initiate this osteoblastic activity. Indeed, the outer callus grows over the intact periosteum and well beyond it until the "unsatisfied end" of the opposite fragment is sealed. The fusiform shape of a callus is not due to a periosteal sheath which connects the bone ends and confines the osteoblastic activity within a fracture hematoma. The callus by-pass is designed to restore continuity by the shortest route possible around the necrotic bone ends. It may be symmetrical if there is an anatomically perfect reduction, and completely asymmetrical if there is bayonet apposition. It is wrong, then, to refer to a periosteal hinge which forms a tube for callus growth; the hinge concept is useful for fracture reduction and immobilization but it is a myth as a sheath or a scaffold for the callus. Indeed, there appears to be a teleologic orientation of the callus which can function independently of any periosteal connection between the fractured ends.

Discussion

The periosteum is a connective tissue sheath or envelope composed of two layers. There is an outer layer which imperceptibly blends with the loose connective tissue beyond it. This fibrous layer is also a pathway for the circumferential and superficial blood supply of the underlying cortex. There is also an inner osteogenic layer of the periosteum. This layer has often been presumed to be the sole source or origin of the so-called periosteal callus. The authors feel that the important osteoblastic cells are found in both layers of

the periosteal sheath, as well as in the parosteal tissue beyond it. In other words, new bone forms underneath, on top of and beyond the periosteum. The specific distribution of this new bone depends on two factors (a) the distribution of the osteoblastic parosteal tissue, and (b) the alignment of the fracture.

Summary

The maturing callus of the rabbit's tibia has been mechanically evaluated, and a graph has been drawn to illustrate the rate of normal healing in a tubular bone. The gradual strengthening of the fracture line has been correlated with gross and microscopical analyses of these calluses. The process of healing has been empirically divided into phases of mobility, plasticity and rigidity, which corresponds roughly to the phases of pre-callus, periosteal callus and endosteal callus. It has been demonstrated that compression does not hasten callus formation in fresh experimental fractures of tubular bones. An attempt has been made to demonstrate that the so-called periosteal callus originates from the entire parosteal tissue and not exclusively from the periosteum: (1) This osteoblastic parosteal tissue is not equally distributed; it is more abundant where bone is covered by muscle, and it is less abundant where bone is subcutaneous. (2) This tissue will form new bone only in contact with live bone, most likely by a process of induction. (3) It will form bone when stimulated by movement.

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Résumé

Dans cet article les auteurs rapportent les résultats de séries d'expériences pratiquées sur des lapins, visant à savoir s'il existe dans une fracture fraîche un moment de solidification particulier et défini. Le rôle des phénomènes de compression a aussi été analysé. Des fractures de la région moyenne de la diaphyse tibiale furent faites de façon à obtenir des traits aussi semblables que possible; ces fractures furent ensuite fixées par des broches type Kirschner, une dans le sens longitudinal et deux transversales, de part et d'autre du trait, ces deux dernières étant bloquées dans le plâtre. La réduction fut contrôlée aux rayons x. Pour mesurer le degré de solidification, les animaux furent sacrifiés; le tibia fut prélevé et après ablation de la broche médullaire on soumit l'os à des essais de traction allant jusqu'à la rupture du cal. Dans ces conditions, des comparaisons sont possibles entre la résistance mécanique d'un cal et son aspect histologique. On a pu ainsi trouver que le cal passe par trois étapes (mobilité, plasticité et solidité) correspondant aux stades histologiques (cal primaire, cal périostique, cal endostéal). Des expériences portant sur l'effet des forces de compression furent aussi faites. Pour cela on procéda de façon identique, mais à l'aide d'une bande élastique, on effectua une compression sur les broches transverses à travers le plâtre. Il est démontré que cette manœuvre n'aide d'aucune façon la consolidation du cal. Il est vraisemblable que le cal périostique naît non seulement du périoste mais encore d'un ensemble de tissus para-osseux; ces tissus sont plus abondants dans les régions profondes, là où l'os est recouvert de muscles. Ce tissu forme de l'os nouveau à condition de se trouver au contact de l'os vivant.

HISTOLOGICAL AND HISTOCHEMICAL EFFECTS OF CORTISONE AND AN ANABOLIC ANDROGEN ON LONG BONES OF YOUNG COCKERELS AND RATS*

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As a part of the connective tissue, bone is affected by the hormones which influence the formation and development of this tissue. Anti-anabolic cortisone-like steroids inhibit the formation of the organic matrix of the growing bone. The fibrogenesis and the synthesis of mucopolysaccharides are inhibited or delayed by these steroids which also produce negative calcium balance. There is, on the other hand, sufficient clinical and experimental evidence that some anabolic androgens have an opposite action to cortisone and may prevent the inhibitory effects of corticoids on bone organic matrix and on mineralization. The effects of steroids on bone formation have been reviewed recently in detail.[11] Work done previously in this laboratory was concerned mostly with some biochemical effects of corticoids and anabolizers on bone matrix. We were able to show that inhibitory effect of cortisone on the metabolism of S^{35}-sulfated polysaccharides in growing bones of cockerels and in healing bones of rats may be prevented or counteracted by some anabolic androgens.[5-10] Cortisone was also found to significantly decrease the tensile strength of the healing fractured bone in rats, and an anabolic androgen, methandrostenolone (Danabol), prevented the post-cortisone alteration in callus strength when given simultaneously with this anti-anabolic steroid.[2, 19] In a recent work on growing bones of cockerels and rats, we showed that cortisone reduced various components of bone, but when the experimental animals were treated with both cortisone and Danabol this did not occur.[11]

It is very important to realize, however, that these results are only significant under certain experimental conditions and depend upon the strain, age, sex and nutritional state of animals studied. Steroids act on various body receptors and the relative intensity of these effects may depend not only on sex and age, but also on the endocrine balance of animals.

Danabol has been extensively investigated in recent years. Although the action of this anabolizer on bone seems evident, the pattern of clinical treatment is not yet established. Particularly delicate seems the problem of the use of this steroid in orthopedic surgery and in pediatrics.[11] It is also apparent that a most convincing test for a clinician would be histological evidence of the effects of anabolizers on bone formation and bone healing.

Histological effects of corticoids on bones have been studied previously in various animal species. Administration of cortisone has resulted in an increased resorption of bone in rats,[3, 12] dogs,[16] rabbits[15] and hens.[17] The general inhibition of matrix formation produced by cortisone has been considered as one of the basic causes of the experimental osteoporosis.[18] The anabolic steroid, Danabol, was found to prevent histological post-cortisone alteration of healing bones, where both steroids were given simultaneously.[2, 19] Other workers studying young growing rats were unable to prevent post-cortisone bone lesions by the use of another anabolizer, Durabolin (19-nor-androstenolone phenylproprionate).[12] However, these series of experiments are not comparable, as both the age of animals and the hormones used were different, and in our studies we dealt with healing callus of fractured bones.

Most studies on the effects of steroids on bone have dealt with growing or mature animals. The possibility of interference with the earlier stages of bone formation has been indicated by studies on embryonic bones. Corticoids were found to inhibit the growth of explanted chick-embryo

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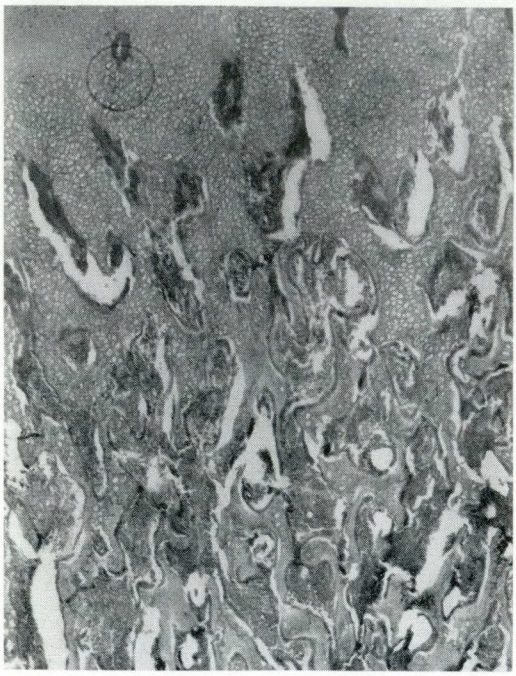

Fig. 1.—Femoral epiphysis of normal four-week-old cockerel showing irregular transition from hypertrophic cartilage (above) to trabeculae of primary spongiosa (below). (H. & E. x 50.)

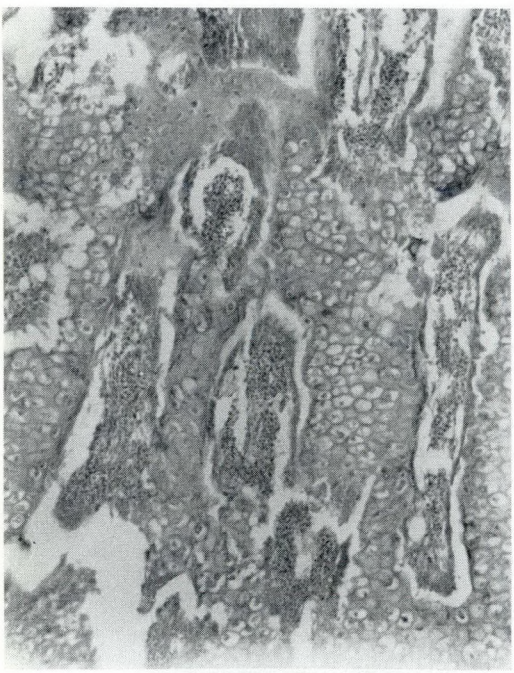

Fig. 2.—Primary spongiosa of normal four-week-old cockerel showing wide trabeculae and presence of nucleated red cells in the marrow spaces. (H. & E. x 125.)

bone anlagen,[1, 4] and this indicated that hormones may indeed influence the early stages of bone formation.

The present report deals with the histological effects of an anti-anabolic steroid, cortisone, and an anabolizer, Danabol, on the long bones of rats and cockerels during the first four weeks after birth.

Method

Young Sprague-Dawley female rats and young cockerels were used.[9] The femoral epiphyses of birds and the humoral epiphyses of rats were studied histologically at the ages of one day, and one, two, three and four weeks. The representatives of anti-anabolic and anabolic steroids, selected for this work, were cortisone (Cortone) and methandrostenolone (Danabol).

Steroids were given subcutaneously, every second day, beginning at the age of seven days in cockerels and at the age of 10 days in rats. Five milligrams of cortisone (0.1 ml.) and 1 mg. of Danabol (0.1 ml.) were the respective doses.

Three groups of cockerels, 30 birds in a group, were treated respectively with Cortone, Danabol and Cortone with Danabol. Ten birds from each group were then killed, one, two and three weeks later, for histological studies. Three groups of rats, 10 animals in a group, had similar injections, but were all killed after two weeks of steroid treatment.

Because of the young age of these animals, decalcification before microtome sectioning was not found necessary. The entire animal was placed in formalin at the time of sacrifice. Femoral bones of cockerels and humeri of rats were then dissected out and placed in formalin. Routine paraffin sections of the entire bone were made. Hematoxylin and eosin stain was used for the study of general morphology, alcian-blue stain[13] for acid mucopolysaccharides and Gomori's stain[13] for alkaline phosphatase.

Results

Cockerels.—At three weeks, routine stains on normal control cockerels revealed a wide band of eosinophilic proliferating cartilage and a thinner band of more basophilic and vacuolated hypertrophic carti-

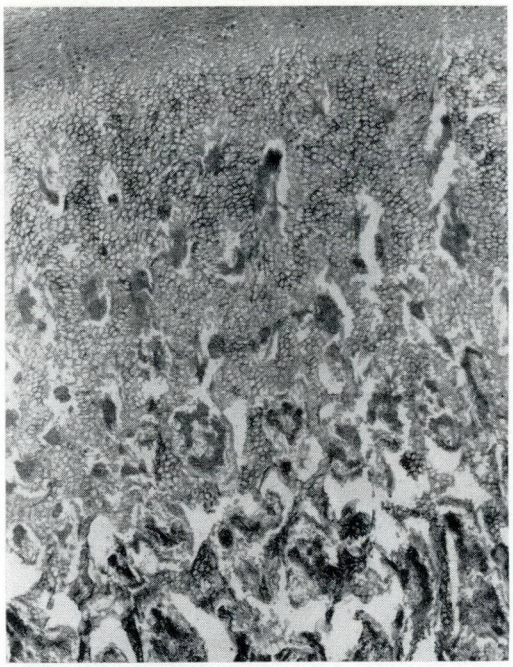

Fig. 3.—Femoral epiphysis of cortisone-treated four-week-old cockerel showing enlargement of epiphyseal cartilage cell lacunae (above) and attenuated trabeculae of primary spongiosa (below). Compare with Fig. 1. (H. & E. x 50.)

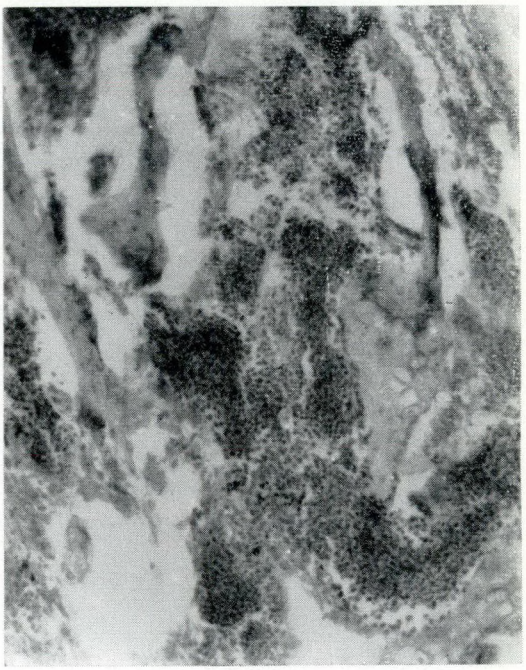

Fig. 4.—Primary spongiosa of cortisone-treated four-week-old cockerel showing considerable loss of trabecular substance. Compare with Fig. 2. (H. & E. x 125.)

lage cells in the epiphysis (Fig. 1). The junction between the hypertrophic cartilage cells and the primary spongiosa formed an interdigitating pattern. The trabeculae in the primary spongiosa varied in thickness from three to 10 cartilage cells (Fig. 2). The outer surfaces of the trabeculae were lined by one to several layers of spindle-shaped to cuboidal osteoblasts with vesicular nuclei and darkly basophilic cytoplasm. The intervening spaces were filled with the usual avian type of nucleated red cells. Fatty tissue in the bone marrow did not appear until the level of the secondary spongiosa.

There were no changes apparent histologically with routine and special stains in the cockerels sacrificed at one and two weeks after injection of cortisone, Danabol, and cortisone and Danabol. In the three-week cortisone-treated group, the width of the various layers did not appear to be remarkably changed. However, the maturest part of the hypertrophic cartilage showed considerably more vacuolation of component cells (Fig. 3). The bony trabeculae of the primary spongiosa were thinner, being

Fig. 5.—Femoral epiphysis of cortisone and Danabol-treated four-week-old cockerel showing no difference from the control. Compare with Fig. 1. (H. & E. x 50.)

not more than three to five cartilage cells in width (Fig. 4). The distribution of osteoblasts was more variable than in the control group, there being patchy areas of almost complete absence. Routine stains in the three-week Danabol-treated cockerels revealed no apparent differences from the control animals. Similarly, the cortisone and Danabol-treated group was not significantly different from the normal (Fig. 5).

In the control cockerels, the alcian-blue stain for mucopolysaccharides resulted in significant staining only along the margins of the chondrocytic lacunae of the proliferating cartilage (Fig. 6). In the hypertrophic zone there was also intense staining of the chondrocytic cytoplasm. This staining gradually disappeared from the trabeculae of the primary and secondary spongiosa concomitantly with replacement of the cartilage cells by osteoid. In the cortisone-treated group decrease in alcian-blue staining was noted largely in the mature part of the hypertrophic cartilage, presumably due to greater vacuolation of the component cells (Fig. 7). Staining of cartilage in trabeculae of the primary spongiosa was not significantly reduced in relationship to the individual cartilage cell but was considerably less because of the decreased width of the trabeculae. No significant differences from the normal control were noted in the Danabol, and the cortisone and Danabol-treated groups.

Alkaline-phosphatase stains in the control cockerels revealed black pigmentation of the osteoblasts lining the trabeculae in the primary spongiosa (Fig. 8). In the cortisone-treated cockerels, pigmentation was only seen in occasional areas where there was persistence of osteoblasts (Fig. 9). However, this was not as prominent as in the controls. No differences from the controls were noted in the Danabol, and in the cortisone and Danabol-treated animals.

Rats.—The normal rat humerus differed considerably from that of the normal cockerel femur during the first month of life (Fig. 10). The rat developed a large area of ossification within the epiphysis itself. At the age of 24 days, the ossification centre represented about one-third of the epiphysis. The junction between the hypertrophic cartilage and the primary spongiosa was a relatively straight line (Fig. 11). The maturest cartilage cells were much more vacuolated than in the cockerel. Cartilage cells were not as prominent in the trabeculae of the primary spongiosa as in the cockerel. The bone marrow of the developing rat was much more cellular and the red cells were not nucleated.

In the rats treated with cortisone for two weeks, the most striking difference from normal was marked suppression of formation of the epiphyseal ossification centre (Fig. 12). In a few of these animals an abortive stage could be seen as a central nidus of enlarged lacunae, but in none was there actual formation of trabeculae. In addition, the demarcation between hypertrophic cartilage and primary spongiosa was not quite as regular and well demarcated as in the control group (Fig. 13). The trabeculae of the primary spongiosa tended to be narrower and more irregular than in the normal rat. There was little difference in the number of osteoblasts between the control and cortisone groups. The Danabol, and the cortisone and Danabol-treated groups showed no differences from the control (Fig. 14).

With alcian-blue stain, the normal rats had much the same pattern as seen in the cockerels with the hypertrophic cartilage taking considerably more stain than the proliferating cartilage. The general intensity of staining throughout was decreased in the cortisone-treated rats. However, no difference from normal was noted in the Danabol, and the cortisone and Danabol groups. With alkaline phosphatase stains, little difference was observed between the control, cortisone, Danabol and cortisone, and Danabol-treated animals.

Discussion

In this experimental study, cortisone effects on long bones of immature rats were present after two weeks of treatment but not until after three weeks in cockerels of the same age. The induced irregularities of trabeculae of the primary spongiosa were the same as described in osteoporosis of older treated animals of both species.[12, 17] However, absence of the normally occurring epiphyseal centre of ossi-

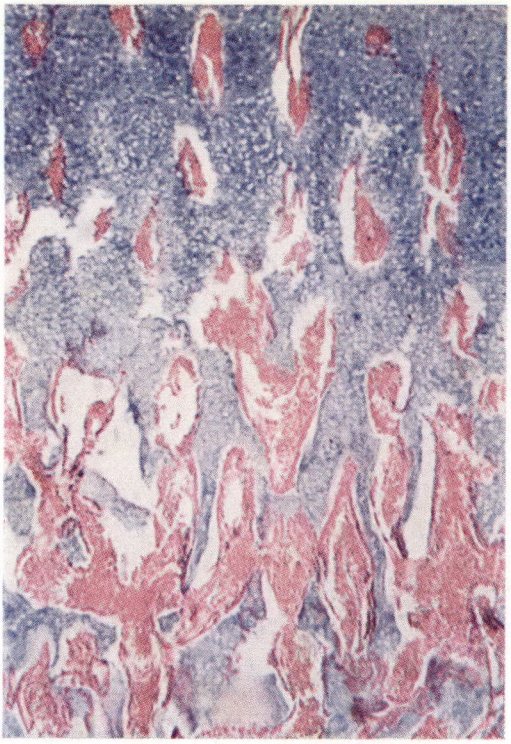

Fig. 6.—Femoral epiphysis of normal four-week-old cockerel stained for acid mucopolysaccharides. The intensity of blue colouration is proportional to the concentration of this substance. Greatest staining is seen in the hypertrophic cartilage (above) with lesser degree in trabeculae of the primary spongiosa (below). (Alcian-blue stain x 50.)

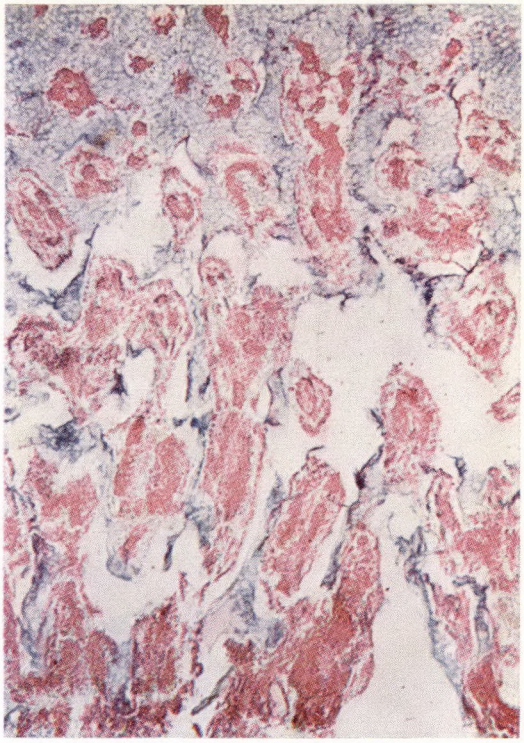

Fig. 7.—Femoral epiphysis of cortisone-treated four-week-old cockerel showing overall decrease in blue staining of acid mucopolysaccharides in hypertrophic cartilage (above) and primary spongiosa (below). Compare with Fig. 6 (Alcian-blue stain x 50.)

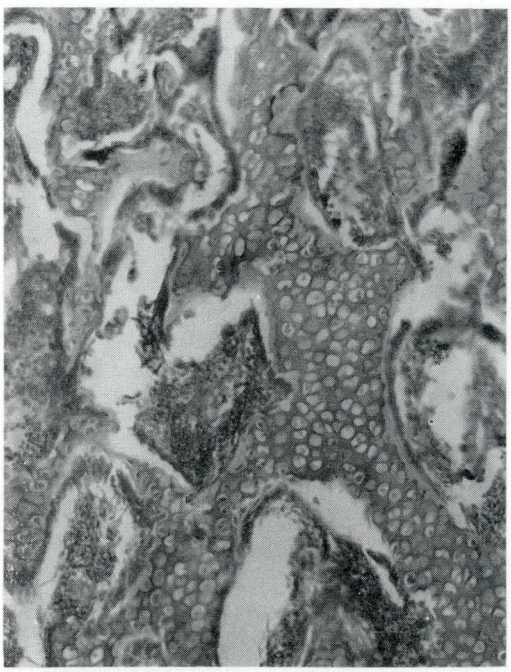

Fig. 8.—Femoral primary spongiosa of normal four-week-old cockerel showing intense reaction for alkaline phosphatase in the lines of osteoblasts along the margins of trabeculae. (Gömöri's alkaline-phosphatase stain x 125.)

Fig. 9.—Femoral primary spongiosa of cortisone-treated four-week-old cockerel showing absence of osteoblasts and of alkaline phosphatase reaction except for one row of osteoblasts (at top). Compare with Fig. 8. (Gömöri's alkaline-phosphatase stain x 125.)

Fig. 10.—Humeral epiphysis of normal 22-day-old rat showing (from above downward) epiphyseal ossification centre, cartilage zone and trabeculae of primary spongiosa. (H. & E. x 50.)

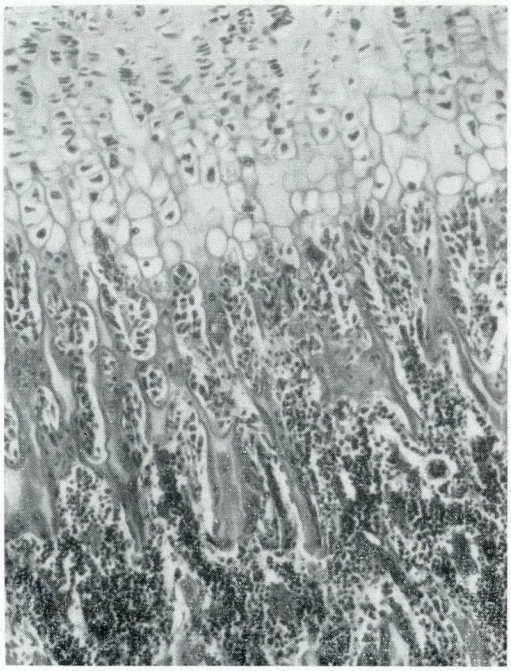

Fig. 11.—Primary spongiosa of normal 22-day-old rat showing regular junction between cartilage (above) and trabeculae (below). (H. & E. x 125.)

Fig. 12.—Humeral epiphysis of cortisone-treated 22-day-old rat showing absence of epiphyseal ossification centre and focus of enlarged lacunae. Compare with Fig. 10. (H. & E. x 50.)

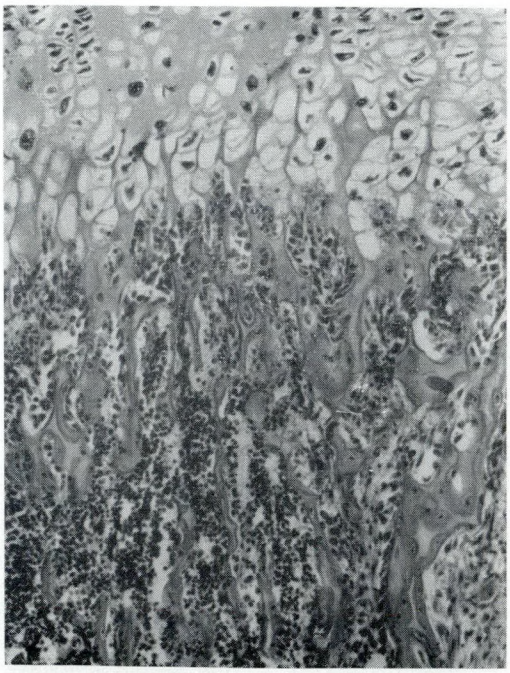

Fig. 13.—Primary spongiosa of cortisone-treated 22-day-old rat showing irregularity of cartilage-trabecular junction and increased size of cartilage lacunae. Compare with Fig. 11. (H. & E. x 125.)

fication in the cortisone-treated rats indicated interference with actual developmental processes during the first month of life. This suggests that the clinical use of cortisone-like steroids during infancy may be a hindrance to maturation of long bones.

The presence of acid mucopolysaccharides, as demonstrated by alcian-blue staining, was decreased in the hypertrophic cartilage and the primary trabeculae of the cockerels and rats treated with cortisone. Decrease in these polysaccharides is indicative of retardation of growth of connective tissue, one of the results of general inhibition of protein synthesis by catabolic steroids such as cortisone.[11]

Tonna and Nicholas[16] have reported sharply reduced alkaline phosphatase activity two to four weeks after autogenous transplants of bone in dogs treated with cortisone. However, it is not clear as to whether the decrease in enzyme activity was due to inhibition of enzyme formation or to the absence of osteoblasts themselves. In the present study, the primary spongiosa of cockerels treated with cortisone had large focal areas in which osteoblasts were absent. With stains for alkaline phosphatase, no reaction was found in these areas, as might have been expected. However, in areas containing osteoblasts, phosphatase activity was substantial. Therefore, it seems likely that the decrease in phosphatase activity with cortisone therapy is merely a secondary result of decreased formation of osteoblasts. This is consistent with the origin of osteoblasts from connective tissue, the growth of which is inhibited by cortisone.

In this series of experiments, some of the histological post-cortisone alterations in growing long bones of rats and cockerels were prevented when cortisone was given simultaneously with the anabolic steroid, Danabol. This agrees with our previous work.[2, 19] In the immature rats used in the present study, not only osteoporosis, but also inhibition of the epiphyseal centre of ossification were obviated in animals treated with both steroids. These findings seem to support a report on counteraction of cortisone-induced bone lesions in humans by administration of some androgenic hormones.[14]

Fig. 14.—Humeral epiphysis of cortisone, and Danabol treated 22-day-old rat showing no differences from the control. Compare with Fig. 10. (H. & E. x 50.)

Summary

The femora of cockerels treated with cortisone during the first four months of life were the site of chondrocytic lacunae of increased size, of thinning and irregularity of primary trabeculae, and of focal absence of osteoblasts. These changes coincided with a decrease in acid mucopolysaccharides and a decrease in alkaline phosphatase. Alkaline phosphatase inhibition was related to a decrease in numbers of osteoblasts.

Somewhat similar changes were seen in the humeri of rats of the same age, except that changes in alkaline phosphatase activity were not evident. In addition, the normally occurring epiphyseal centre of ossification seen in control rats was not present after two weeks of cortisone treatment. No differences from the normal controls were observed histologically in birds and rats treated with Danabol. However, Danabol protected the bones of birds and rats from the deleterious effects of cortisone, when both steroids were given simultaneously.

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Résumé

Le tissu osseux, étant un type spécial de tissu conjonctif, se trouve sujet aux influences hormonales capables d'agir sur le conjonctif. Les auteurs ont étudié ici l'action de la cortisone sur des os d'animaux de laboratoire. Pour leurs expériences, ils utilisèrent des rats ou de jeunes coqs. Les fémurs des poulets traités à la cortisone pendant les premiers quatre mois de leur vie montrèrent une augmentation de la taille des lacunes chondrocytaires, un amincissement et des irrégularités dans les trabécules osseuses. A ces modifications morphologiques correspond une diminution de la teneur en mucopolysaccharides acides et de la phosphatase alcaline. Ce dernier phénomène est probablement dû à la raréfaction des ostéoblastes. Des résultats semblables ont été obtenus dans les humérus de jeunes rats; cependant les modifications de l'activité de la phosphatase alcaline y sont moins marquées. Le centre d'ossification épiphysaire que l'on trouve normalement chez les animaux-témoins, n'apparaît pas après un traitement de deux semaines à la cortisone. Chez les rats ou les poulets traités au Danabol, ces modifications histologiques n'ont pas été retrouvées; de plus, il semble que le Danabol soit capable de protéger le tissu osseux contre les effets nocifs de la cortisone, lorsque ces deux stéroïdes sont administrés simultanément. Les auteurs attirent l'attention sur le fait que les résultats ne sont donnés que pour les espèces animales considérées et que ces résultats peuvent varier selon la race, la souche, le sexe, l'âge et l'état de nutrition des animaux. L'équilibre endocrinien de l'organisme joue également un rôle important dans la réponse aux stéroïdes.

SURGICAL TECHNIQUE

THE BIFRONTAL APPROACH TO ANEURYSMS OF THE ANTERIOR COMMUNICATING ARTERY*

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THE treatment of ruptured intracranial aneurysms is still a matter of some difference of opinion between physician and surgeon and, indeed, between surgeons! It is apparent that surgery must be shown clearly to be superior to fasting and prayer before it can be accepted as the treatment of choice. For the more accessible aneurysms in the better-risk patients this superiority has already been demonstrated.[1] However, surgical treatment of some aneurysms, particularly those of the middle cerebral trifurcation and the anterior communicating artery, is often followed by a high mortality and serious morbidity. Among surgeons there is some difference of opinion regarding the best type of surgical treatment, for example, ligation of the carotid artery in the neck,[2] intracranial clipping of the main feeding artery proximal to the aneurysm,[3] trapping it with clips on either side, or clipping its neck. However, there can be little doubt that the ideal method is one by which the lesion is obliterated, all possibility of recurring hemorrhage is removed and the normal cerebral arterial supply is unaltered. It is our purpose here to emphasize some technical improvements in the direct surgical management of aneurysms of the anterior communicating artery which have helped us to approach this goal.

Botterell *et al.*[4] have emphasized the value of temporary interruption of the intracranial circulation during exposure and clipping of ruptured aneurysms. They further demonstrated the degree of protection against cerebral anoxia from prolonged partial ischemia provided by general body cooling. In April 1960, Pool[5] discussed the advantages of a bilateral frontal approach to the region of the anterior communicating artery, and the use of temporary clips on the arterial trunks intracranially to prevent re-rupture of aneurysms during their exposure. Hypothermia is an essential adjunct to this technique, and the intravenous administration of urea is helpful in obtaining greater brain shrinkage and more room to work. Pool's results were sufficiently impressive that we adopted his method and have used it since in cases of aneurysms in this region.

TECHNIQUE

A bifrontal free bone-flap craniotomy is performed using endotracheal anesthesia, intravenous urea and total body cooling to 30° C. The sagittal sinus is sectioned, and both frontal lobes lifted from the orbital roofs with self-retaining brain retractors (Fig. 1). The olfactory nerves are sacrificed. The internal carotid arteries are cleared of arachnoid and exposed to their bifurcation (Fig. 2). The anterior cerebral arteries are in turn exposed medially to the anterior communicating junction. We have not found it necessary to place temporary clips on the internal carotid arteries in every case. Temporary clips are placed on both anterior cerebral arteries; the anterior communicating artery with the aneurysm is then carefully but fully exposed, rolling the latter forward out of its bed in the anterior wall of the third ventricle. With the aneurysm thus fully exposed, its oozing ruptured dome is seen and the exact relationships of the anterior cerebral arteries are visualized before placing a clip across the base of the aneurysm. The Mayfield spring clip appears to have several advantages, not the least of which is application by relaxation of the operator's hand rather than by the usual squeezing motion which is so often accompanied by slight but dangerous tremor. The temporary clips on the anterior cerebral arteries are then relaxed *in situ*

*From the Division of Neurosurgery, University of Alberta Hospital, Edmonton, Alta.

TABLE I.—Ten Patients with Aneurysms in the Region of the Anterior Communicating Artery**

Case No.	*Age and sex*	*Onset*	*Blood pressure*	*Pre-operative days*	*Pre-operative state*	*Duration of circulatory occlusion*	*Post-operative course*	*Days in hospital*	*Final state*
1	56 F	Unconscious for 12 hours	175/100	5	Drowsy, irrational (2)	0	L. Hemiparesis	102	L. Hemiparesis
2	56 M	Unconscious for 16 hours	130/75	6	Stuporous (2)	45 min.	Confused 3 weeks	26	Labouring 3 mo. after operation*
3	42 M	Headache. Unconscious for 1 hour	140/80	2	Confused (2)	7 min. Interfrontal hematoma	Confused 2 weeks	23	Clerking 2 mo. after operation*
4	44 M	Headache— 3 days	150/100	8	Rigid neck (1)	11 min.	Normal	20	Business 6 wks. after operation*
5	58 M	Headache—24 hrs. Unconscious for 1 hour	170/100	0	Stuporous (3)	12 min. Intracerebral hematoma	Confused 3 weeks	30	Gardening 7 wks. after operation*
6	37 F	Headache—24 hrs. Convulsion and coma—2 hrs.	140/80	1	Coma, flaccid subhyaloid (5) hemorrhages	0 Intracerebral and ventricular hemorrhage	Stupor 5 weeks Tracheotomy	180	Euphoric. Poor vision. Paresis Rt. leg
7	49 F	Headache. Vomiting for 24 hours	165/100	1	Drowsy (2)	9 min. Intracerebral hemorrhage	Frontal lobe syndrome— 5 weeks	36	Housewife 2 mo. after operation*
8	56 F	Blackouts and apathy for 6 months	145/70		Apathetic (1)	0 Artery clipped	Confused. Apathetic	60	Pre-op. mental state 2 mo. after operation*
9	40 F	Unconscious and maniacal—3 hrs.	138/80	0	Coma (4)	13 min. Ventricular hemorrhage	Confused—6 wks. Decerebrate 3rd post-op. day	28	Housewife 2 mo. after operation*
10	55 M	Headache and convulsions for 3 weeks	160/90	21	Confused (4) Spastic legs Subhyaloid hemorrhages	17 min.	Spastic legs. Fatal hemorrhage 7th post-op. day	7	Died from recurrent hemorrhage

** See explanatory details in text.
* Normal.

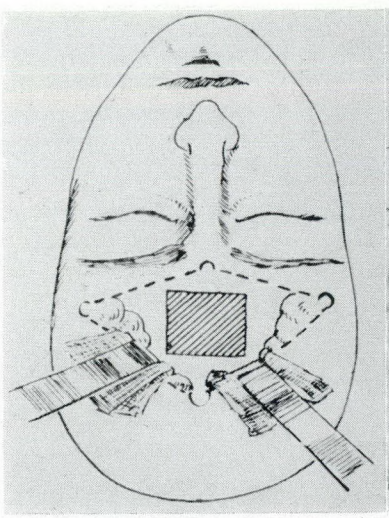

Fig. 1

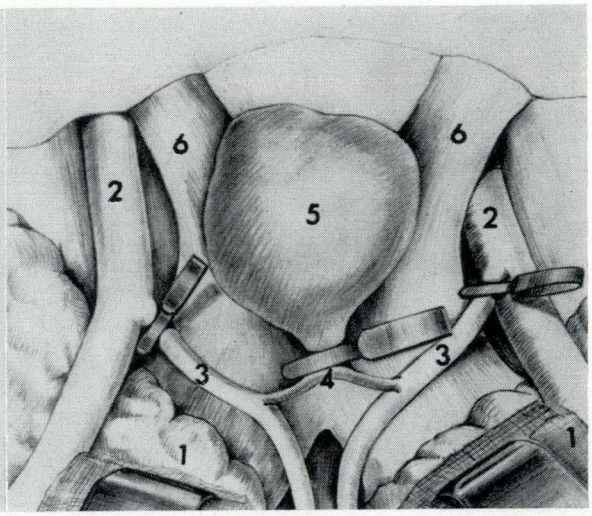

Fig. 2

Fig. 1.—Orientation diagram of the surgical exposure as seen by the surgeon standing above and behind the patient's head. The shaded area is shown in enlarged detail in Fig. 2.
Fig. 2.—Details of the surgical exposure of the chiasmal region after retraction of the frontal lobes (1). The internal carotid arteries (2) are cleared of arachnoid to their bifurcations into middle and anterior (3) cerebral arteries. The latter are then occluded by temporary clips and the anterior communicating artery (4) with the aneurysm (5) arising from it is exposed, taking care to avoid injury to the optic nerves (6) and chiasm immediately subjacent. The neck of the aneurysm is then clipped permanently, avoiding any obstruction to the distal portion of the anterior cerebral arteries.

seriatim, observing the aneurysm for further leaking and the anterior cerebral arteries distal to the aneurysm for good filling, indicating that normal circulation has been restored. Closure is carried out taking special care to cover the usually widely opened frontal sinuses with a flap of pericranium sutured to the dura.

Case Material

Ten patients with an aneurysm of the anterior communicating artery were treated in this manner by the authors at the University of Alberta Hospital between May 1960 and January 1962 (Table I). Nine of these aneurysms had ruptured before operation, while one (Case 8) had produced a "frontal lobe syndrome" owing to pressure on the medial surface of the frontal lobes and interference with the anterior cerebral artery circulation. One other patient with a ruptured anterior communicating artery aneurysm was seen during this period but the lesion was not amenable to surgical treatment by this technique. That patient was decerebrate and in acute pulmonary edema on admission and subsequently became akinetic and mute. Despite this, operation was performed five days later when he suffered

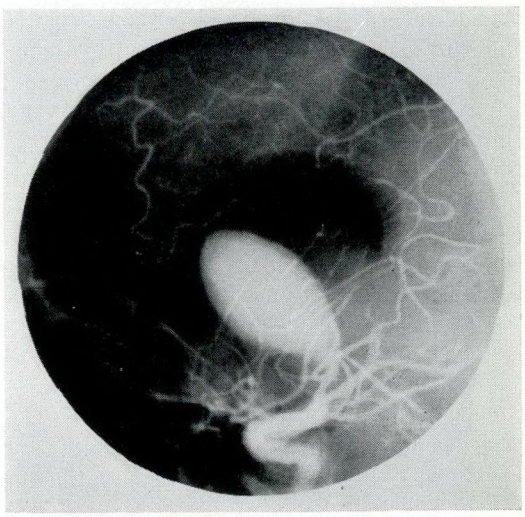

Fig. 3.—The combined preoperative pneumoencephalogram and carotid angiogram from Case 8, the only unruptured aneurysm in this group. It was subsequently clipped and removed by the bifrontal approach, but circulation in the distal anterior cerebral arteries was not improved in the postoperative angiograms.

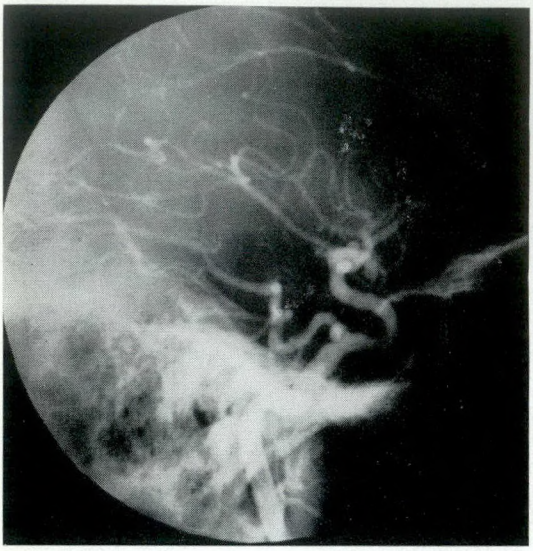

Fig. 4

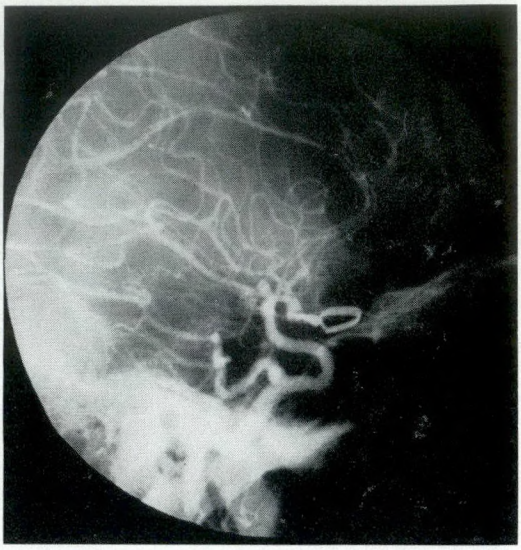

Fig. 5

Figs. 4 and 5.—Preoperative and postoperattive angiograms of a patient (Case 7) with a trigeminal artery as well as a ruptured anterior communicating artery aneurysm. Note the apparent aneurysmal dilatation of the origin of the trigeminal and middle cerebral arteries.

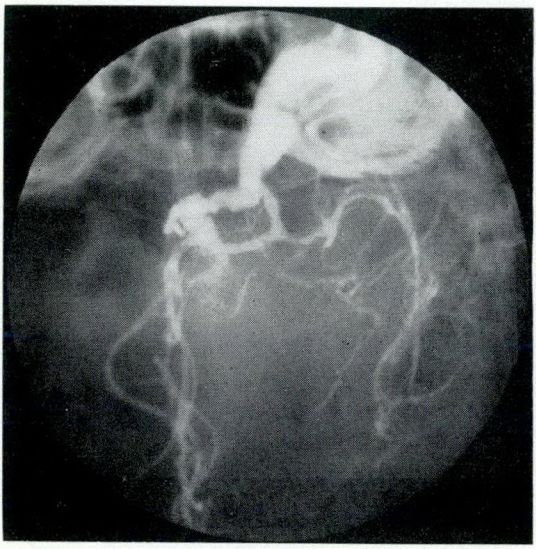

Fig. 6

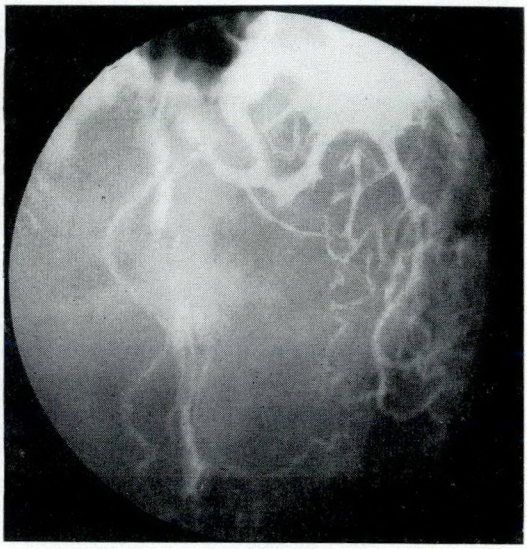

Fig. 7

Figs. 6 and 7.—Preoperative and postoperative angiograms from Case 10. Note the position of the clip across the body of the aneurysm in the postoperative film, instead of at the base of the aneurysm where it had been placed at operation. The significance of this was not realized until the patient died of recurrent hemorrhage seven days after operation and two days after postoperative angiography, and the clip was found to have been "pumped off" the aneurysm.

a further hemorrhage. A massive intracerebral hemorrhage and edema made the Pool approach impossible, and this patient, therefore, is not included in this series. With this exception, all patients with aneurysms in this area seen by the authors during this period were subjected to operation as soon as possible after diagnosis.

Results

The details regarding the individual patients are recorded in Table I and are discussed below.

Preoperative Days

The figures in this column refer to the time between the subarachnoid hemorrhage and operation. The longer periods are due to the patient's initial admission to other hospitals, frequently at some distance. It is our practice to treat all cases of ruptured intracranial aneurysms as surgical emergencies once the patients are admitted to our care.

Preoperative State

The bracketed numbers in this column are the grading of the patients immediately before operation according to the Botterell criteria.[4] It will be seen that this group of patients is representative, varying from the best to the worst surgical risks, a matter of some significance in relation to the end results.

Duration of Anterior Cerebral Artery Occlusion

Temporary occlusion of both anterior cerebral arteries varied from 0 to 45 minutes. It was our impression that adequate hypothermia will protect the frontal lobes from the effects of this type of temporary partial anoxia for at least the maximum (45 minutes) recorded here. It should be noted that the circulatory arrest is only partial, and is variable from patient to patient, depending upon the back-flow through communicating branches from the middle cerebral arteries. As noted in the description of the technique, there is usually sufficient communication to allow the ruptured aneurysm to bleed a little, even after clipping of both anterior cerebral arteries.

The presence of significant intracerebral, intercerebral and intraventricular clots is also recorded in this column.

Postoperative Course

It is clear that the immediate postoperative course is more closely related to the preoperative state and the finding of intracerebral clots and brain destruction than it is to the duration of temporary anterior cerebral artery occlusion.

Final Results

The longest follow-up period for any patient in this group is now two years and the shortest, nine months. It will be seen that seven patients have returned to their normal occupation and habits of life, while two are seriously handicapped and one is dead. The latter patient (Case 10) died on the seventh postoperative day from recurrent hemorrhage when the clip was "pumped off" the aneurysm (Fig. 7). Of the two handicapped, one (Case 1) is the result of an unexplained postoperative thrombosis of the right middle cerebral artery. This vessel was not exposed or interfered with in any way at operation; in fact there was no temporary occlusion of any vessel during operation upon this patient. In no case have we seen evidence of damage or thrombosis at the site of temporary clips in the routine follow-up angiograms.

Discussion

This technique offers two important advantages: (1) Improved exposure of the area, and (2) prevention of premature rupture of the aneurysm during exposure.

1. *Improved exposure of area.*—The excellent exposure of this area from both sides, as well as from in front and above is obvious from the artist's sketches (Figs. 1 and 2). It results directly from the bifrontal flap and sectioning of the falx and sagittal sinus, but is augmented by the use of intravenous urea and hypothermia.

2. *Prevention of premature rupture of the aneurysm during exposure.*—Protection against a re-rupture of the aneurysm as it is being exposed is afforded by the use of temporary clips, which in turn are only safe when hypothermia is employed to protect the brain from the effects of anoxia. To be able to separate an anterior communicating aneurysm carefully from the adjacent brain or optic chiasm, to identify clearly its relationships to the anterior cerebral arteries, and to apply the clip across its neck without muss or fuss is an experience which should be enjoyed by every neurosurgeon.

The disadvantages peculiar to this method of surgical exposure are: (1) The wide opening of the frontal sinuses which increases the possibility of ascending infection or cerebral spinal fluid fistula formation, and (2) the sacrifice of the olfactory nerves. We have had no serious difficulties from either of these sources. All of the patients received antibiotics for from five to seven days after their operation. Only two patients in this series have demonstrated minor evidence of infection, one six weeks after operation and another almost one year postoperatively. Two of our patients have mentioned an awareness of the loss of the sense of smell.

It was of some interest to review the angiograms in these 10 patients. Although no concerted effort was made to demonstrate cross-filling, five patients showed filling of the aneurysm, as well as both anterior cerebral arteries, from one side. This finding was verified at operation when, in each instance, only a tiny or vestigial first portion of anterior cerebral artery was present on the other side. These observations lead us to suspect that the treatment by proximal clipping of the anterior cerebral artery[3] in many of our patients would have led to ischemia and infarction in both anterior cerebral artery distributions.

It is clear from what has been said previously that we believe immediate operation is indicated for most patients suffering from ruptured intracranial aneurysms if they are fit to stand an operation. In this group we would include many who are comatose, so long as no definite sign of major structural brain damage is present, as indicated by Cases 5, 6, 9 and 10. Many patients are comatose from the moment of their subarachnoid hemorrhage, but recover full consciousness and function within 12 to 24 hours. One can only speculate on how many die from a preventable second hemorrhage when operation is delayed in the unconscious patient because of undue consideration for "statistics" based on the same sort of selection.[6] Such figures cannot take into account preventable recurrent hemorrhages during the unconscious period.

One final point of interest concerns the two patients who had gross intraventricular hemorrhage. Both were treated by ventricular lavage at the time of operation; one (Case 6) has severe physical impairment as a result of cerebral and ocular damage. The other (Case 9) made a complete recovery despite decerebrate episodes on the third and fourth postoperative days. We suspect these were the result of intermittent intraventricular obstruction from residual clots. It may be that routine ventricular puncture is indicated during operation where the aneurysm may have ruptured into the ventricular system. If gross blood and blood clot are present, ventricular lavage may prevent some postoperative deaths.

Summary

Ten patients with aneurysms in the region of the anterior communicating artery were treated by a method which provides complete and almost bloodless exposure of the lesion and vessels involved, and avoids the surgical chaos of attempting to clip an actively bleeding aneurysm before it and the adjacent vessels have been completely exposed and protected. The preoperative condition of these patients varied from Grade I to Grade V according to the Botterell classification. They were operated upon as early as possible after diagnosis. Nine of the patients survived operation and seven have returned to full normal activity.

Conclusions

The bifrontal approach to the region of the anterior communicating artery fulfills the first principle in surgery — that of adequate exposure, which the authors have been unable to obtain heretofore by the unilateral subfrontal or midline approaches. The clipping of aneurysms of this vessel is further facilitated by the use of intravenous urea to shrink the brain, and hypothermia to permit temporary interruption of the circulation. The end-results in this small series hold promise of improvement in the mortality and morbidity in the management of this lethal lesion.

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Résumé

Le traitement des anévrysmes intra-crâniens est encore très discuté. Ceux notamment qui intéressent la trifurcation cérébrale moyenne et l'artère communicante antérieure sont responsables d'un taux de mortalité très élevé. Du point de vue chirurgical, on a proposé soit la ligature de l'artère carotide interne au niveau du cou, soit le clippage de l'artère principale anévrysmée à l'intérieur du crâne. Les auteurs rapportent et discutent ici quelques pratiques techniques qu'ils ont mises au point. Sous anesthésie générale et hibernation à 30° C., on pratique une craniotomie bifrontale. Le sinus sagital est sectionné et les deux lobes frontaux sont soulevés à l'aide de rétracteur spéciaux; les nerfs olfactifs sont sacrifiés. On a alors accès sur les artères carotides internes ainsi que leurs branches et les artères cérébrales antérieures qui sont clampées temporairement; on expose l'anévrysme de l'artère communicante que l'on clippe. Dix malades ont été soumis à cette intervention au "University of Alberta Hospital". Les résultats sont: un décès, deux patients gravement diminués et sept bien portants, ayant repris leurs activités normales depuis au moins neuf mois. Les auteurs insistent sur le fait que ces bons résultats sont avant tout dus à une très bonne exposition des lésions à traiter.

ANATOMY IN SURGERY. 2nd ed. Philip Thorek. 904 pp. Illust. J. B. Lippincott Company of Canada Ltd., Montreal, 1962. $23.75.

The evaluation of this second edition of a widely known book poses a dilemma for the reviewer. It is obviously an important book without being a distinguished one; it is highly useful without being invaluable; it is a labour of love, but it does not engender complete respect. The very size of the author's undertaking and the degree of success that he achieves deserve admiration; but some of his methods and the decisions which underlie these methods do not.

Like its predecessor, this edition claims that it does not stress surgical techniques; nevertheless, descriptions of surgical procedures occupy a substantial part of the text. In such passages the author intends the stress to be on the anatomy that underlies the procedure. Often he succeeds in this. However, he fails in carrying out his design sufficiently often for it to be noticeable.

"Anatomy in Surgery" is successful when viewed from the special point of view of the surgical resident. Because he finds that most of its contents are of practical use to him, the resident hardly cares whether the book is a hybrid. He is unlikely to attach much importance to the criticism, legitimate in the reviewer's approach, that much of the anatomical description is pedestrian and unoriginal.

The resident will be greatly impressed by the profusion of good illustrations, a regrettably large number of which appear to be unacknowledged copies from standard textbooks such as J. C. B. Grant's Atlas of Anatomy (e.g., Figs. 451, 514 and 565). Such carping criticisms will not reduce the book's usefulness to the novice. On the other hand, frequent errors and laxity can be harmful. These include errors in spelling (e.g. "Descement's" membrane) and in the use of unorthodox terms (e.g., spinal "medulla" for spinal cord), the use of many passages that can have only the slightest practical interest to the surgeon (e.g., "The average weight of the human brain in the adult male is approximately 1380 grams; of the female, approximately 1250 grams . . ."), and finally, errors in fact or concept (e.g., the stylomandibular ligament in Fig. 92).

This reviewer cannot help but admire the author's industry and singleness of purpose. Dr. Thorek has succeeded in producing the book he obviously set out to produce. It is a book that will attract and hold the attention of almost any surgeon-in-training in a way that more scholarly publications cannot hope to achieve.

CANADIAN JOURNAL OF SURGERY

All communications concerning this Journal should be marked "Canadian Journal of Surgery" and addressed to the Editor, C.M.A. Publications, at C.M.A. House, 150 St. George St., Toronto 5.

The Journal is published quarterly. Subscription is $10 per year ($5 per year for trainees in surgery), and starts with the January issue of each year. Single copies are $2.50 each, payable in advance. (It would be greatly appreciated if subscribers would please add bank exchange to their cheques.)

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Manuscripts **in duplicate** of original articles, case reports, and other contributions should be forwarded with a covering letter requesting consideration for publication in the *Canadian Journal of Surgery*. Acceptance is subject to the understanding that they are submitted solely to this Journal, and will not be reprinted without the consent of the author and the publishers. Acceptance or rejection of contributions will be determined by the Editorial Board. As space is available, a limited number of case reports will be published. Articles should be typed on one side only of unruled paper, double-spaced, and with wide margins. The author should always retain a carbon copy of material submitted. Every article should contain a summary of the contents. The Concise Oxford Dictionary will be followed for spelling. Dorland's American Medical Dictionary will be followed for scientific terminology. The Editorial Board reserves the right to make the usual editorial changes in manuscripts, including such changes as are necessary to ensure correctness of grammar and spelling, clarification of obscurities or conformity with the style of the *Canadian Journal of Surgery*. In no case will major changes be made without prior consultation with the author. Authors will receive galley proofs of articles before publication, and are asked to confine alterations of such proofs to a minimum.

Reprints

Reprints may be ordered on a form which will be supplied with galley proofs. It is important to order these before publication of the article, otherwise an extra charge for additional type-setting will be made.

References

References should be referred to by numerals in the text. They should include in order: the author's name and initials in capitals; title of the article; abbreviated journal name; volume number, page number and year. The abbreviations of journal names should be those used by the National Library of Medicine, Washington, D.C., as published in *Index Medicus*. References to books should include in order: author's name and initials; title of book; number of edition (e.g., 2nd ed.); title of publishing house; city of publication; year of publication; page number if a specific reference.

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A reasonable number of black-and-white illustrations will be reproduced free with the articles. Colour work can be published only at the author's expense. Photographs should be glossy prints, unmounted and untrimmed, preferably not larger than 10″ x 8″. Prints of radiographs are required and *not the originals*. The magnification of photomicrographs must always be given. Photographs must not be written on or typed on. An identifying legend may be attached to the back. Patients must not be recognizable in illustrations, unless the written consent of the subject for publication has been obtained. Graphs and diagrams should be drawn in India ink on suitable white paper. Lettering should be sufficiently large that after reduction to fit the size of the Journal page it can still be read. Legends to all illustrations should be typed separately from the text and submitted on a separate sheet of paper. Illustrations should not be rolled or folded.

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It should be clearly understood that contributors are at full liberty to submit articles in either English or French, as they please. Acceptance will be quite independent of the language of submission. If the contributor wishes, he may submit an informative summary of not more than 300 words in the language other than that in which he has submitted the article. For example, an article in English must carry an English summary and may, if the author wishes, carry a more detailed summary in French.

The Royal College of Physicians and Surgeons of Canada

NEWSLETTER

REVIEW OF FELLOWSHIP EXAMINATIONS

From the floor of the Annual Business Meeting in Edmonton in January came an expression of concern regarding the apparent increasingly high failure-rate among candidates for the College Fellowship examinations. After citing figures to support his statement, Dr. Carman Weder of Saskatoon, seconded by Dr. C. T. Wolan also of Saskatoon, presented a motion calling for the establishment of a committee, broadly representative of the geographic, language and age-group distributions within the present Fellowship in the College, to define the objectives of the Fellowship examinations and to recommend how these objectives might best be attained.

Dr. Ronald V. Christie, Chairman of the Committee on Examinations, reported that that Committee also had been concerned at the high failure-rate resulting from the Fellowship examinations in recent years. At its meeting in December 1962, three subcommittees had been established to carry out a searching review of the whole structure and content of the Fellowship examinations in Medicine, Surgery and Obstetrics and Gynecology. Depending on the findings of the review of these three major specialties, it was likely that the examinations in other specialties would be subjected to a similar review.

In view of the study already being undertaken by the Committee on Examinations, Dr. Weder and Dr. Wolan agreed to withdraw their motion in favour of one moved by Dr. E. M. Nanson of Saskatoon, directing that the findings and conclusions of the study by the Committee on Examinations be circulated to the Fellows and presented at the next Annual Business Meeting. The motion was duly seconded and carried.

"PILOT STUDY" OF EVALUATION OF TRAINING PROGRAMS

For some years the Committee on Approval of Hospitals has been concerned about the means at its disposal for obtaining an adequate qualitative assessment of graduate training-programs in hospitals approved or seeking approval by the College for resident training. While general principles and prerequisites and certain quantitative criteria for approval have been established for most of the specialties and are readily assessable, there are less tangible factors which may have an important influence on the quality of training. The Committee believes that these factors can only be properly evaluated through on-the-spot visits by persons thoroughly knowledgeable and experienced in the field of graduate medical education.

The chief impediment to the undertaking of a program of first-hand reviews of graduate training programs by qualified individuals designated by the College has been the sizable expenditure which such a program is likely to involve. However, the Council recognizes that in placing its stamp of approval on training programs, there is an inherent responsibility for assuring trainees that such programs provide a well-organized training of high standard, designed to prepare them adequately for the practice of their specialty and for the examinations of the College. For this reason, Council believes it must give careful attention to possible alternative ways and means of improving its assessment of training programs and institutions.

The "pilot study", which was approved by Council at its January meeting, will endeavour to determine the value of on-the-spot evaluation of training programs, as well as the administrative problems and costs involved. The study will embrace

a review of the graduate training programs in all of the approved hospitals and institutions in the London area, and will be carried out by a team consisting of Dr. Robert Kerr, Professor of Medicine at the University of British Columbia, and Dr. Donald R. Webster, formerly Chairman of the Department of Surgery and now Director of the Department of Experimental Surgery at McGill University. Both have a wide knowledge and experience in graduate medical education which will enable them to exercise a considered judgment on the organization, quality and effectiveness of training programs. In addition, both are members of the Council of the College and of the Committee on Approval of Hospitals and, therefore, are thoroughly acquainted with and understand the objectives of this study.

The activities of the College in prescribing training requirements and in approving centres for graduate-training in the clinical fields often impinge on areas of immediate interest and concern to university medical schools and the teaching hospitals. A lack of knowledge and of appreciation of College objectives has occasionally led unwittingly to the creation of problems or misunderstandings between these interested groups. At the suggestion of the Association of Canadian Medical Colleges, the Council, at its January meeting, approved the establishment of a Joint Committee representing the Royal College, the A.C.M.C. and the university teaching hospitals. Through this Joint Committee closer liaison and communication between the three groups will be possible and future difficulties and misunderstandings avoided.

REVISIONS TO TRAINING REQUIREMENTS

At its January meeting, Council gave approval to a single standard of training requirements for both the Fellowship and Certification examinations in the specialties of Diagnostic Radiology, Therapeutic Radiology and Urology. The revised requirements, which must be fulfilled by candidates for the examinations in these specialties commencing in 1967, will be published shortly in the various medical journals. Candidates presently in the course of training or about to embark upon training in these specialties and who expect to appear for the examinations in 1967 or later, may obtain copies of the revised requirements by writing to the College office.

The Council also approved a minor change in the uniform training requirements in Plastic Surgery which were published in the *Canadian Journal of Surgery* in October 1962. The change provides for the inclusion of General Surgery among the options for the year of training under section 2(b) of the new requirements. This section will now read as follows:

(b) One year of approved resident training in surgery, to include training in at least two of the following: general surgery, neurosurgery, orthopedic surgery, otolaryngology and urology.

REVISIONS TO CONDITIONS FOR ANNUAL MEDAL AWARDS

The Committee on Annual Awards has expressed disappointment in the rather limited number of essays submitted in recent years in competition for the annual Medal Awards in Medicine and Surgery of The Royal College of Physicians and Surgeons of Canada. The Committee is convinced that a great deal more work is being done in Canada which would qualify for consideration for these awards than is presently being submitted to it. Because of this, the Committee has recommended and Council has approved a number of changes in the conditions attached to these awards, which it is hoped will serve to stimulate a greater interest in and competition for the awards. Most important among the changes are:

—The age limit for those who may apply for the awards has been extended from 41 to 45 years as at December 31 of the year in which the work is submitted.

—Authors will be required to submit a manuscript in a form suitable for publication and, in addition, an abstract of not more than 300 words. In the judging of submissions, considerable reliance will be placed on the scientific merit of the work as described in the abstract, and less emphasis given to

the literary quality and neatness of preparation of the manuscript.

—All abstracts submitted will, in addition to being considered for the Medal Awards, be considered by the Program Committee for the Annual Meeting for presentation in the regular scientific sessions of the Annual Meeting.

—The deadline for submission of manuscripts and abstracts will be September 30, the same as that for the offers of papers for the Annual Meeting program.

—The Medal Award will be accompanied by a cash prize of $500 to the recipient, in place of the present policy of paying his expenses to the Annual Meeting.

Because of the implications of the above changes for candidates who may already be well advanced in the preparation of manuscripts for the awards this year under the old terms and conditions of the awards, the above revisions will become applicable to submissions made in 1964 for the 1965 Awards.

1963 REGIONAL MEETING

The 1963 Regional Meeting of the College will be an Eastern Ontario Regional Meeting to be held at Etherington Hall, Queen's University, Kingston, on Thursday, Friday and Saturday, October 3, 4 and 5, 1963.

Mailed invitations to attend this Regional Meeting have been sent to all Fellows and Certificated Specialists in Eastern Ontario and Western Quebec, although the meeting will be open to any specialist or interested medical practitioner throughout the Provinces of Ontario and Quebec.

Enquiries concerning this meeting and offers of papers for the scientific program should be directed to Dr. H. Garfield Kelly, Chairman of the Program Committee, Etherington Hall, Stuart Street, Kingston, Ont.

IMPORTANT 1963 VISITORS

The 1963 McLaughlin-Gallie Visiting Professor will be Professor Lance Townsend, Professor of Obstetrics and Gynecology at the University of Melbourne, Australia. Professor Townsend will be in Canada for approximately two months commencing about September 1. His major visits will be to the University of Toronto and to Dalhousie University.

The 1963 Sims Commonwealth Travelling Professor to Canada is Mr. C. Naunton Morgan, Surgeon to St. Bartholomew's Hospital, London, England. Mr. Morgan will also be in Canada for approximately eight weeks, commencing in September, and will pay brief visits to a number of university centres in both Eastern and Western Canada.

It is hoped that both Professor Townsend and Mr. Morgan will be able to participate as guest lecturers at the Regional Meeting in Kingston.

W. Gordon Beattie, F.R.C.S.[C],
Honorary Assistant Secretary,
March 4, 1963.

1962 FELLOWSHIP EXAMINATIONS

The names of the surgical candidates who were successful in the 1962 Fellowship examinations of the Royal College of Physicians and Surgeons of Canada are listed below.

GENERAL SURGERY

Ainslie, William Hugh, M.D. (Toronto) 1954, Niagara Falls, Ont.

Beauregard, Pierre, M.D. (Montréal) 1957, Montreal, Que.

Capello, Peter Joseph, M.D. (Ottawa) 1956, Montreal, Que.

Chaikof, Leo, M.D. (Toronto) 1954, Downsview, Ont.

Briere, Jacques, M.D. (Montréal) 1957, Montreal, Que.

Collins, David Larkham, M.D., C.M. (McGill) 1954, Vancouver, B.C.

Durrani, Khalid Mahmud Khan, M.B., B.S. (Punjab) 1954, Detroit, Mich., U.S.A.

Field, Paul, M.B., B.S. (London) 1954, Sudbury, Ont.

Fortin, Claude Lucien, M.D. (Laval) 1958, Quebec, Que.

Friesen, Gerhard, M.D. (Manitoba) 1951, Winnipeg, Man.

Gendron, Joseph Edmond, M.D. (Toronto) 1950, Cobourg, Ont.

Hache, Lorenzo, M.D. (Ottawa) 1957, Montreal, Que.

Hodgins, Thomas Emerson, M.D. (Western Ontario) 1951, Sarnia, Ont.

HOLUBITSKY, ISADORE BORIS, M.D. (Alberta) 1955, Vancouver, B.C.
KERR, WILLIAM HUDSON, M.D. (Toronto) 1956, Don Mills, Ont.
KING, JOHN KENNEDY, M.D. (Toronto) 1954, Grimsby, Ont.
LAVOIE, DANIEL, M.D. (Laval) 1954, Montreal, Que.
LAVOIE, JOSEPH GUY PIERRE, M.D. (Montréal) 1958, Rosemont, Montreal, Que.
MAMMEN, KANDATHIL OOMMEN, M.B., B.S. (Madras) 1955, Madras, India.
MARTYN, JOHN WALTER, M.D. (Toronto) 1954, R.R. 3, Peterborough, Ont.
MAYBA, IHOR IVHEN, M.D. (Manitoba) 1957, Winnipeg, Man.
MCDOUGALL, EDWARD PETER, M.D. (Toronto) 1956, Toronto, Ont.
MCINTYRE, JOHN ADDISON, M.D. (Toronto) 1951, Uxbridge, Ont.
MOREAU, ANTONIO, M.D. (Montréal) 1956, Montreal, Que.
ORT, REGINALD ROSS, M.D. (Western Ontario) 1957, London, Ont.
PELOQUIN, JOSEPH BERNARD ANDRE, M.D. (Montréal) 1958, Montreal, Que.
PEREY, BERNARD JEAN FRANCOIS, M.D. (McGill) 1956, Montreal, Que.
POISSON, ROGER CELESTIN, M.D. (Ottawa) 1956, Montreal, Que.
RAWLING, EDWARD GEORGE, M.D. (Western Ontario) 1957, London, Ont.
REINGOLD, MARVIN, M.D. (Toronto) 1955, Downsview, Ont.
RICHARDSON, PETER, M.B., B.S. (Durham) 1954, Regina, Sask.
SCHARF, ROBERT FRANKLIN, M.D., C.M. (McGill) 1948, Ottawa, Ont.
SCOTT, DAVID JOHN, M.D. (Toronto) 1956, Orangeville, Ont.
SHANDLING, BARRY, M.B., Ch.B. (Cape Town) 1950, Toronto, Ont.
SIEGENBERG, JOE, M.B., Ch.B. (Johannesburg) 1953, Edmonton, Alta.
SIWAK, WALTER JOHN, M.D. (Alberta) 1951, Camrose, Alta.
SLADEN, JOSEPH GILBERT, M.D. (Toronto) 1954, Toronto, Ont.
TEWARSON, IVAN PURUSHOTTAM, M.B., B.S. (Madras) 1953, Bombay, India.
VAIL, WILLIAM JAMES, M.D. (Ottawa) 1952, Canadian Army, Germany.
WHITE, HUBERT CARMAN, M.D. (Toronto) 1956, Chatham, Ont.
WILKINSON, ROBERT HERBERT, M.D. (Toronto) 1955, Weston, Ont.
YOUNG, JOHN KEITH, M.D. (Alberta) 1954, Edmonton, Alta.

SURGERY (CARDIOVASCULAR AND THORACIC SURGERY)

LEPAGE, GILLES, M.D. (Montréal) 1951, Ville Mont-Royal, Montreal, Que.

SURGERY (NEUROSURGERY)

HARDY, JULES, M.D. (Montréal) 1956, Montreal, Que.
HUESTIS, WILLIAM STUART, M.D., C.M. (Dalhousie) 1956, Halifax, N.S.
MURRAY, JAMES KENNETH, M.D. (Toronto) 1953, Burlington, Ont.

SURGERY (OBSTETRICS AND GYNECOLOGY)

BATE, JOHN THOMAS, M.D. (Toronto) 1957, Weston, Ont.
BRODY, HARRY, M.D. (Alberta) 1956, Calgary, Alta.
CATTERILL, THOMAS BRIAN, M.D., C.M. (McGill) 1954, Montreal, Que.
DAY, RICHARD ALLAN, M.D. (Toronto) 1957, Edmonton, Alta.
DOERFFER, FREDERICK ROBERT, M.D., C.M. (Queen's) 1946, Hamilton, Ont.
DORR, HUMPHREY PATRICK, M.D., C.M. (McGill) 1956, Montreal, Que.
EISEN, ISADORE MURRAY, M.D. (Toronto) 1951, Toronto, Ont.
EISTETTER, ALFRED EDWARD, M.D. (Western Ontario) 1955, Saskatoon, Sask.
GOODWIN, JAMES WILLIAM, M.D. (Toronto) 1955, Edmonton, Alta.
HARVEY, JAMES MILN, M.D. (Toronto) 1957, Toronto, Ont.
IRWIN, KARL MAXWELL, M.D. (Toronto) 1955, Toronto, Ont.
REID, DAVID WILLIAM JAMES, M.D. (Alberta) 1956, Edmonton, Alta.
TOWELL, MOLLY ETHEL, M.B., B.S. (London) 1952, Vancouver, B.C.
TRUEMAN, EVELYN GRACE TESS, M.D. (Alberta) 1953, Calgary, Alta.
WAXMAN, BENNY, M.D. (Western Ontario) 1957, Arlington, Va., U.S.A.
WINCH, GUY CAMERON, M.B., Ch.B. (St. Andrew's, Scotland) 1952, North Vancouver, B.C.

SURGERY (OPHTHALMOLOGY)

HARRIS, GORDON SHEFFIELD, M.D. (Toronto) 1952, R.R. 1, Terra Cotta, Ont.
HASSARD, DONALD THOMAS ROBERTSON, M.D. (Alberta) 1957, Edmonton, Alta.
KROLMAN, GORDON MARSHALL, M.D. (Manitoba) 1955, Winnipeg, Man.

PRATT-JOHNSON, JOHN ASHBURNHAM, M.B., B.Ch. (Witwatersrand) 1951, Vancouver, B.C.

ROUSSEAU, ALAIN PAUL, M.D. (Laval) 1956, Ste-Foy, Quebec 10, Que.

SURGERY (ORTHOPEDIC SURGERY)

BLANCHARD, GUY ANDRE, M.D. (Ottawa) 1956, Duvernay, Montreal, Que.

CLOUTIER, JEAN-MARIE, M.D. (Ottawa) 1954, Montreal, Que.

ELLIS, CHRISTOPHER FRANCIS GEORGE, M.D. (Western Ontario) 1956, Montreal, Que.

EMERY, MICHAEL ALBERT, M.D., C.M. (McGill) 1957, Edmonton, Alta.

GROVE, NED MURRAY, (formerly GROSSBERG), M.D. (Toronto) 1957, Chicago, Ill., U.S.A.

HENDERSON, MURRAY CUYLER, M.D. (Western Ontario) 1955, Burlington, Ont.

RICHTER, ROBERT ROY, M.D. (Toronto) 1947, Hamilton, Ont.

TRIAS, ANTONI, M.D. (Barcelona) 1952, Armdale, N.S.

WATT, JOHN GORDON, M.D. (British Columbia) 1956, Richmond, B.C.

WILSON, DAVID WILMOT, M.D. (Toronto) 1955, Washington, D.C., U.S.A.

SURGERY (OTOLARYNGOLOGY)

BRODOVSKY, DAVID MARTIN, M.D. (Manitoba) 1957, Winnipeg, Man.

SURGERY (PLASTIC SURGERY)

COURTEMANCHE, ALBERT DOUGLAS, M.D. (Toronto) 1955, Vancouver, B.C.

NICOLLE, FREDERICK VILLENEUVE, M.B., B.Ch. (Cambridge) 1956, Montreal, Que.

WALKER, FREDERICK GEORGE, M.D. (Alberta) 1956, R.R. 1, Ottawa, Ont.

SURGERY (UROLOGY)

HYAMS, BRAHM BALLON, M.D., C.M. (McGill) 1956, Montreal, Que.

KERESTECI, ARA KAPRIEL, M.D. (Istanbul) 1954, Toronto, Ont.

LAPALME, ROGER, M.D. (Montréal) 1957, Montreal, Que.

SALES, JACK LEROY, M.D. (Western Ontario) 1955, Thamesville, Ont.

SMYLIE, DONALD THOMAS, M.D., C.M. (Queen's) 1959, Ottawa, Ont.

TODD, IAIN ALEXANDER DRYSDALE, M.B., B.Ch. (Cambridge) 1956, Don Mills, Ont.

TY, MANUEL, JR., M.D. (Philippines) 1952, Saskatoon, Sask.

ORTHCOMING MEETINGS

THE SOUTH WESTERN ONTARIO SURGICAL ASSOCIATION

The Annual Meeting of The South Western Ontario Surgical Association will be held in the Busby Memorial Auditorium, Victoria Hospital, London, Ont., on Wednesday, November 20, 1963.

For further information, write to Dr. Robert M. McFarlane, Secretary, The South Western Ontario Surgical Association, Surgery Office, Victoria Hospital, London, Ont.

THE CANADIAN SOCIETY FOR THE STUDY OF FERTILITY

The Tenth Annual Meeting of the Canadian Society for the Study of Fertility will take place at The Canadiana Motor Hotel, Highway 401 and Kennedy Road, Agincourt, Toronto, Ont., on June 12 and 13, 1963.

For further information, please write to the Secretary, Dr. George H. Arronet, Infertility Centre, Royal Victoria Hospital, Montreal, P.Q.

EYE SURGERY CLINICAL MEETING

An Eye Surgery Clinical Meeting, sponsored by the Division of Postgraduate Medical Education, has been arranged by the Department of Ophthalmology in the University of Toronto for Thursday and Friday, June 6 and 7, 1963.

Professor G. Meyer-Schwickerath, M.D., Director of the Essen City Hospital Eye Clinic, Essen, Germany, and Mr. James R. Hudson, M.B., F.R.C.S., D.O.M.S., Ophthalmic Surgeon, Moorfields Eye Hospital, London, England, will be the guest speakers.

Surgical clinics will be held in the teaching hospitals in the mornings, and in the afternoons the registrants will be divided into groups and will attend short demonstrations and seminars on selected surgical subjects. The University staff will take part in the afternoon seminars and demonstrations.

The fee for the Clinical Meeting is $35.00 (Canadian funds). Application should be made to the Director, Division of Postgraduate Medical Education, Faculty of Medicine, University of Toronto, Toronto 5, Ont., before May 15, 1963.

NOTICES

CANADIAN FELLOWS OF THE AMERICAN COLLEGE OF OBSTETRICIANS AND GYNECOLOGISTS

The American College of Obstetricians and Gynecologists recently announced the granting of Fellowship in the College to the following residents of Canada: Joseph B. Boulos, St. John's, Newfoundland; Jacques Corbeil, Marcel Ferron, Constant Nucci, Oscar Nutik, Percy H. Roberts, Morris Sabin, Jean-Paul Thibault, H. Maurice Weisberg and Albert E. Wolfe, all of Montreal; Gerald Beaudry, Sherbrooke, P.Q.; Harley J. Hughes, Fort William, Ont.; Fred L. Johnson and Gerard J. Quigley, Hamilton, Ont.; Kadri Ulman, Kingston, Ont.; Robert B. Gibb, Kitchener, Ont.; Norman Edward Miller, Sault Ste. Marie, Ont.; Wm. G. Francis and Milton Raymers, Toronto; Leo Kawula and K. B. Lazarus, Regina, Sask.; Albert B. Brown, Saskatoon; Norris R. Bertrand and John E. McAllister, Calgary, Alta.; James R. Leeder, Edmonton, Alta.; and Roland W. Radcliffe, Nanaimo, B.C.

PROFESSOR IAN AIRD

Mr. Hugh McLeave, the author of Sir Archibald McIndoe's biography, has been invited by the family of the late Professor Ian Aird to write his official biography which will be published by Messrs. William Heinemann Ltd.

Mr. McLeave would welcome the contribution of any letters or papers written by Professor Aird, or any anecdotes concerning his life and work. All documents of this nature will be returned to the sender.

Material contributed for this purpose should be directed to Mr. Hugh McLeave, 15 Ladbroke Grove, Holland Park, London, W.11, England.

Books Received

Books are acknowledged as received, but in some cases reviews will also be made in later issues.

Accident Surgery. Vol. I. Edited by H. Fred Moseley. 242 pp. Illust. Appleton-Century-Crofts, New York, 1962. $10.00.

An Atlas of Prostatic Surgery. Perry B. Hudson and Arthur Purdy Stout. 170 pp. Illust. W. B. Saunders Company, Philadelphia; McAinsh & Co. Ltd., Toronto and Vancouver, 1962. $14.60.

Automation of the Microbiological Assays of Antibiotics with an Auto-Analyzer Instrumental System. Andres Ferrari, John R. Gerke, Thomas A. Haney, M. E. Madigan, Joseph F. Pagano. Annals of the New York Academy of Sciences. Vol. 93, Art. 13, pages 625-648. Illust. Editor: Mary C. Johnstone. New York Academy of Sciences, New York, August 20, 1962.

Buerger's Disease. A Follow-Up Study of World War II Army Cases. Michael E. DeBakey and Bernard M. Cohen. 143 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1963. $9.25.

Comparative Virology. Conference Editor: H. Koprowski. Annals of the New York Academy of Sciences. Vol. 101, Art. 2, pages 327-582. Editor: Mary C. Johnstone. New York Academy of Sciences, New York, November 30, 1962.

Conference on Clean Surfaces with Supplement: Surface Phenomena in Semiconductors (Symposium). Conference Editor: F. R. Eirich. Annals of the New York Academy of Sciences. Vol. 101, Art. 3, pages 583-1014. Illust. Editor: Mary C. Johnstone. New York Academy of Sciences, New York, January 23, 1963.

Control of Infections in Hospitals. Hospital Monograph Series No. 12. J. C. Colbeck. 166 pp. American Hospital Association, 840 North Lake Shore Drive, Chicago 11, Ill., 1962. $2.00.

Current Diagnosis and Treatment. Henry Brainerd, Sheldon Margen, Milton J. Chatton and associate authors. 842 pp. Lange Medical Publications, Los Altos, Calif., 1963. $9.50.

Endogenous Metabolism with Special Reference to Bacteria. Consulting Editor: Carl Lamanna. Annals of the New York Academy of Sciences. Vol. 102, Art. 3, pages 515-793. Illust. Editor: Harold E. Whipple. New York Academy of Sciences, New York, January 21, 1963.

Fat Embolism. Simon Sevitt. 233 pp. Illust. Butterworth & Co. (Canada) Ltd., Toronto, 1962. $13.50.

The Hemorrhagic Disorders. 2nd ed. Mario Stefanini and William Dameshek. 614 pp. Illust. Grune & Stratton, Inc., New York; The Ryerson Press, Toronto, 1962. $23.75.

Hospital Administration. Geoffrey A. Robinson. 480 pp. Butterworth & Co. (Canada) Ltd., Toronto, 1962. $15.75.

Hypothermie: de la physique à la physiopathologie per-opératoire (Hypothermia: From Physiology to Preoperative Physiopathology). L. Vadot. 126 pp. Illust. L'Expansion Scientifique Française, Paris, 1962. 26 NF. $5.20 (approx.).

Les Ictères (The Varieties of Jaundice). Guy Albot et Félix Poilleux, et collaborateurs. 345 pp. Illust. Masson & Cie, Paris, 1962.

Introduction biochemique à la chirurgie. Marcel Florkin. 248 pp. Illust. Masson & Cie, Paris, 1962. 38 NF. $7.60 (approx.).

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BOOK REVIEWS

(See also pages 140, 160, 186, 217 and 243)

INTRAVENOUS CHOLANGIOGRAPHY. Robert E. Wise. With a foreword by Richard B. Cattell. A monograph in American Lectures in Roentgen Diagnosis, edited by Lewis E. Etter. 139 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1962. $10.50.

Dr. Wise has written a personal and very lucid account of seven years' experience with intravenous cholangiography. Most readers will be surprised to learn that visualization of the bile ducts was obtained in almost 90% of cases. This, no doubt, is due to careful selection of patients and good radiographic technique, of which tomography is the cornerstone. The causes of non-visualization may be summarized as liver damage, increasing jaundice or biliary fistula. Thus, such examination is often successful when jaundice is receding.

Two significant points arise from the study. The fact that the author found no evidence that the common bile duct dilates after cholecystectomy, and his enunciation of the "time-density-retention" principle in the diagnosis of common duct obstruction, consideration of which will result in an 86% accuracy in diagnosis.

The criteria for the application of the time-density-retention principle are as follows:

Delayed opacification of the bile ducts with increasing density at 120 minutes. Additional signs are: lack of tapering of the common bile duct; dilatation of hepatic duct radicles; and, if a previous examination has been performed, increasing size of the common bile duct.

Pitfalls in diagnosis are considered in some detail, and these range from air bubbles and layering of dye to false gallbladder shadows produced by the duodenum or gastric antrum; Fig. 49 is an excellent example of the latter.

Non-visualization of the gallbladder in the presence of adequate visualization of the bile ducts indicates gallbladder disease, with the qualification that in about 10% of cases it is due to inspissated bile caused by pancreatic or primary common duct disease. In the diagnosis of pancreatic disease only 50% accuracy is claimed but, on occasion, the intravenous cholangiogram may anticipate diagnosis by other methods.

Some minor criticisms may be advanced. There is confusion as to the number of examinations performed, these being variously quoted as 2028, 2032 and 2034. Fig. 37 is not a convincing example of the point that the author is attempting to make. The reviewer does not share the author's enthusiasm for polaroid prints which, with some exceptions, were not as good as the conventional ones.

This is an excellent book which is of particular interest to the radiologist, but it is also highly recommended to the surgeon and internist.

TREATMENT OF CANCER AND ALLIED DISEASES. Vol. 5. Tumors of the Gastrointestinal Tract, Pancreas, Biliary System, and Liver. 2nd ed. Edited by George T. Pack and Irving M. Ariel. 828 pp. Illust. Paul B. Hoeber, Inc., New York, 1962. $33.00.

This well-illustrated volume deals in considerable depth with all aspects of intra-abdominal malignant disease. It will find its greatest use in the hands of the practising surgeon and advanced surgical trainee who requires detailed information on the anatomical, physiological and clinical problems inherent in the care of patients with malignant disease in this situation. It is not basically designed for students. The accepted diagnostic and classical features of the various diseases receive brief but adequate coverage.

The book is divided into one introductory section on general principles of treatment and five regional sections. The latter include sections on neoplasms of the stomach; of the small intestine, mesentery and retroperitoneum; the pancreas, biliary system and liver; the colon, and the rectum and anus.

The various chapters have been contributed by 68 recognized authorities, largely drawn from American centres. As a result, uniformity of subject presentation has not been achieved and, of necessity, some repetition occurs. However, in this particular field, the documentation of different approaches to management is probably desirable. Where appropriate, collected statistical data are recorded for ease of comparison.

Extended radical procedures for the management of gastric and colonic cancer receive detailed discussion. Liver neoplasms, both primary and secondary, are dealt with extensively. The relatively uncommon malignant lesions of the mesentery and peritoneum receive more thorough consideration than in most other contemporary texts. Palliative treatment, including irradiation, and newer developments in ancillary care are discussed fully. The inclusion, throughout the book, of sections devoted to minor problems of frequent occurrence in the surgical and postsurgical phase of the management of such patients is a refreshing innovation since such topics receive scant mention in most texts on the subject.

This book will appeal particularly to those practising surgeons who are primarily concerned with and deeply interested in the problem of the management of patients with intra-abdominal cancer.

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STEREOENCEPHALOTOMY. Part II. Clinical and Physiological Applications. E. A. Spiegel and H. T. Wycis. 504 pp. Illust. Grune & Stratton, Inc., New York; The Ryerson Press, Toronto, 1962. $29.75.

The authors of this volume were among the earliest workers in human stereotaxic surgery and their contribution to this field is second to none on this continent. The monograph will serve as a monument to their efforts in this respect and will provide a useful reference for students to the historical background, methods and results of this technique of brain surgery. Unfortunately, the rapid changes which have been made in this field in the past few years have rendered large sections of the book almost obsolete. In addition, this reviewer gains the impression that at least one of the reasons for the collection of all this material (much of which has been published previously) into one volume has been to emphasize the pre-eminence of the authors' work.

The clinical problems that Spiegel and Wycis have treated with stereoencephalotomy include schizophrenia, Parkinson's disease, hemiballismus, athetosis, torsion spasm, intractable pain, petit mal and subcortical tumours. The quantity of their case material is small, as one would anticipate in a procedure where much time was required for exploration and development. The case history abstracts and the results reported do not in themselves constitute a major therapeutic triumph, but certainly bear witness to the surgical courage and investigative enthusiasm of the authors. One cannot help but feel that their efforts might have been more productive had they been able to explore one field thoroughly before going on to another. The monograph itself would have been improved by better organization and a more concise style. It runs to over 500 pages, including the index, and over one-third is devoted to a discussion of destruction of the dorsal medial nucleus of the thalamus for mental disease. Since this is really of only historical interest now and has been dealt with in previous publications, it could have been omitted entirely or very briefly summarized.

Of special interest is the detailed discussion on the development of the stereotaxic technique used by the authors, and their evaluation of the variability of the various structures within the thalamus.

The graduate student and practising neurosurgeon interested in stereoencephalotomy will find this volume a valuable introduction to the historical background, basic anatomy and problems of design of human stereotatic devices. It leaves something to be desired in so far as the discussion of clinical case material and the evaluation of results is concerned.

THORACIC AND CARDIOVASCULAR SURGERY WITH RELATED PATHOLOGY. 2nd ed. Gustaf E. Lindskog, Averill A. Liebow and William W. L. Glenn. 1024 pp. Illust. Appleton-Century-Crofts, New York, 1962. $18.00.

This second edition of a text first published in 1953 has been increased by the addition of a large section on cardiovascular surgery. The first section on lung surgery gives a satisfactory coverage and in itself is a good reference text. However, the reviewer receives the impression that some of the details of treatment have not been brought up to date. There is a good historical review in each chapter and, of course, much of the treatment is of historical interest also. In the cardiovascular section, the various congenital and acquired lesions are discussed fully along with the present techniques and equipment. In such a rapidly changing specialty, it is difficult to keep really up to date, but the authors have made a very satisfactory effort.

NOUVELLE PRATIQUE CHIRURGICALE ILLUSTREE. Fascicule XIX (New Surgical Practice Illustrated, Fascicle XIX). Edited by Jean Quénu. 295 pp. Illust. G. Doin & Cie, Paris, 1962. 52 NF. $10.40 (approx.).

Tout l'intérêt de ce volume de 280 pages, réside dans la manière succincte, mais suffisamment explicative qu'on emploie, pour exposer le cas clinique en cause, et dans les excellentes illustrations de P. Rivallain qui, à elles seules, deviennent un véritable traité de technique chirurgicale.

Les auteurs n'ont fait le choix que de cas de pratique courante, relevant ou bien d'une entité pathologique établie, ou bien d'une complication d'actes chirurgicaux antérieurs.

Le lecteur a donc ici le choix d'un groupe de lésions chirurgicales nécessitant un traitement que les auteurs ont essayé de déterminer à l'avance.

Par ailleurs, la nature et l'extension des lésions est telle dans quelques cas, que la technique, si bien figurée, a dû être adaptée aux circonstances anatomiques, inflammatoires ou tumorales et représente de ce fait un apport nouveau si on les compare à celles que contiennent les traités habituels.

La rectopexie discal, et la greffe *in situ* dans le mal de Pott lombaire, nous paraissent des traitements intéressants et nouveaux. Cependant, nous entretenons une certaine inquiétude en regard de la dégénérescence possible et de l'amincissement du disque L^5 S^1 dans la rectopexie, mais l'auteur n'en fait aucunement mention.

C'est enfin un volume extrêmement intéressant.

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NOUVELLE PRATIQUE CHIRURGICALE ILLUSTREE. Fascicule XX (New Surgical Practice Illustrated, Fascicle XX). Edited by Jean Quénu. 284 pp. Illust. G. Doin & Cie, Paris, 1962. 50 NF. $10.00 (approx.).

Les techniques chirurgicales décrites dans ce volume continuent pour nous à présenter le même intérêt que les fascicules précédents. Voyons ensemble ce que semble présenter d'intéressant chacune de ces interventions.

La première nous traite de la "Commissurotomie mitrale au dilatateur".

La contribution de Christian Cabrol à cette intervention, consiste dans une manipulation différente des anticoagulants avant et après l'opération mais surtout dans l'usage du dilatateur de Dubost.

Le second article traite de la "Pleurectomie pariétale pour pneumothorax idiopathique".

M. Claude Dubost met ici en pratique l'idée de Gaensler, de supprimer la cavité pleurale par pleurectomie pariétale. Le cas présenté permet une belle étude de la technique opératoire et démontre la possibilité d'une complication redoutable, l'hémorragie.

Dans une troisième opération présentée par M. Jean Loygne, on peut repasser ici "l'Opération de Heller pour Méga œsophage idiopathique".

C'est un bel exposé d'une méthode éprouvée depuis plusieurs années, l'œsophago-cardiomyotomie. Elle est poussée ici le plus possible, et un soin extrême est mis à la section des fibres musculaires du cardia.

La fermeture de l'angle de His est ajoutée, pour éviter le reflux œsophagien.

Dans un article suivant, de M. Claude Dubost, sur l'hépatico-jéjunostomie en Y, l'intérêt réside ici: 1° dans la lésion méconnue lors d'une première intervention chirurgicale; 2° dans la dissection nécessitée par les réactions inflammatoires locales; 3° par les anastomoses en un plan qu'on voit de plus en plus utilisées. Résultat qui semble parfait.

Dans la "Greffe de la bifurcation aortique pour artérite chronique des membres inférieurs", M. Georges Thomeret illustre parfaitement les difficultés immédiates comme les thromboses, et éloignées comme la circulation plus difficile qu'auparavant, avec claudication, pieds froids, douleurs.

Le prochain article traite de la "Cure opératoire d'un grand diverticule vésical". Ici, M. Louis Quénu nous donne un conseil qui semble bien utile, c'est de modifier légèrement la technique de Marion pour ouvrir la vessie en latérale, près du collet diverticulaire, et une fois le diverticule réséqué, il n'y a plus qu'une ouverture vésicale à suturer. Beaucoup moins de délabrement.

Enfin, M. Paul Pandovani, nous apporte dans la "Cure opératoire des luxations tarso-métatarsiennes, par réduction et arthrodèse", une intéressante contribution au traitement d'une lésion qui guérit mal par la simple immobilisation. La fixation par les broches des os luxés, donne un résultat remarquable.

Il faut souligner ici l'excellente contribution qui se continue d'ailleurs des figures faites par M. P. Rivallain si précieuses à l'exposé de la technique.

ATHLETIC INJURIES. Prevention, Diagnosis and Treatment. 5th ed. Augustus Thorndike. 260 pp. Illust. Lea & Febiger, Philadelphia; The Macmillan Company of Canada Limited, Toronto, 1962. $5.50.

This book, in its fifth edition, is still simple and concise. For this reason, it will continue to have a considerable appeal for both medical and non-professional personnel responsible for the welfare of athletes.

The section on statistics has been improved by the addition of new cases and by a longer follow-up period. The number of illustrations has not been substantially increased but a few have been replaced by better examples. Finally, the bibliography has been brought up-to-date.

The publishers should be commended for an excellent production.

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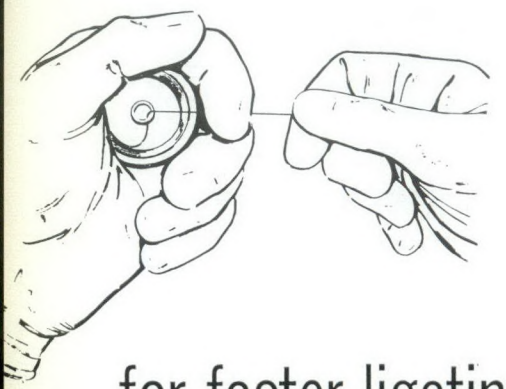

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True radiopacity of DAVIS & GECK DISPENSER REEL is shown at (1). The reel was taped to the abdomen and the exposure was made. Metal eyelet renders reel radiopaque.

Davis & Geck Surgical Gut Dispenser Reel

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(Continued from page 254)

CORRELATIVE NEUROANATOMY AND FUNCTIONAL NEUROLOGY. 11th ed. Joseph G. Chusid and Joseph J. McDonald. 386 pp. Illust. Lange Medical Publications, Los Altos, Calif., 1962. $5.50.

With the passage of time and reproduction through 11 "generations", this useful little handbook, once called *Correlative Neuroanatomy*, has grown to the adult proportions of the "compleat" neurological treatise. It has, in fact, become a textbook of neurology that stresses the neuroanatomical background of disease. It is still very useful and easy to read, and contains excellent sketches, illustrations and concise discussion. However, it need no longer be considered a supplement to standard neurological texts. True, it lacks the learned discussions and detailed references to the literature by which students are so often confused, but it contains nearly all that is clinically useful in neuroanatomy, neurophysiology, neurology, neurosurgery and neuroradiology. It can therefore be recommended as *the* basic textbook to be bought by the student in first-year medicine and used constantly throughout his medical course, and even in postgraduate study (outside the specialty fields of neurology). Used in that fashion, it will become an invaluable handbook for all practitioners, and a repository for much useful information which so easily escapes the mind. However, the steadily increasing size of this once modest and particularly useful synopsis of neuroanatomy is noted with regret.

PLASTIC AND RECONSTRUCTIVE SURGERY OF THE EYE AND ADNEXA. Papers presented to the First International Symposium of the Manhattan Eye, Ear and Throat Hospital. Edited by Richard C. Troutman, John M. Converse and Byron Smith. 305 pp. Illust. Butterworth & Co. (Canada) Ltd., Toronto, 1962. $8.50.

This book is a collection of the papers presented to the First International Symposium of the Manhattan Eye, Ear, Nose and Throat Hospital on plastic and reconstructive surgery of the eye and adnexa.

To the ophthalmologist, the first two sections dealing with general principles of tissue transplantation, foreign implants and general plastic surgical techniques will prove interesting. The remainder of the book is as disappointing as the symposium itself. There is little, if anything that is new and not already fully covered in standard ophthalmological textbooks and in articles in ophthalmological journals. The papers written in foreign languages are very sketchily summarized in English and some are not translated at all.

The acquisition of this book will add little to the ophthalmologist's library. A plastic surgeon who may have had little experience in this field would be better advised to purchase a standard textbook on the subject.

(Continued from page 251)

Local Analgesia: Abdominal Surgery. 2nd ed. Sir Robert Macintosh and R. Bryce-Smith. 82 pp. Illust. E. & S. Livingstone Ltd., Edinburgh & London; The Macmillan Company of Canada Limited, Toronto, 1962. $4.50.

Meteoritic Hydrocarbons and Extraterrestrial Life. Edward Anders. Discussion by Bartholomew Nagy, Warren G. Meinschein and Douglas J. Hennessy. Annals of the New York Academy of Sciences. Vol. 93, Art. 14, pages 649-664. Illust. New York Academy of Sciences, New York, August 29, 1962.

Methotrexate in the Treatment of Cancer. Report of the Proceedings of a Symposium at the Royal Society of Medicine, 18 September, 1961, sponsored by Lederle Laboratories, a Division of Cyanamid of Great Britain Ltd., Chairman: Prof. F. Bergel. Edited by Ruth Porter and Eve Wiltshaw. 80 pp. Illust. John Wright & Sons Ltd., Bristol; The Macmillan Company of Canada Limited, Toronto, 1962. $5.85.

Modern Trends in Neurology. Third Series. Edited by Denis Williams. 384 pp. Illust. Butterworth & Co. (Canada) Ltd., Toronto, 1962. $17.00.

Modern Trends in Orthopaedics. Third Series. Fracture Treatment. Edited by J. M. P. Clark. 259 pp. Illust. Butterworth & Co. (Canada) Ltd., Toronto, 1962. $13.50.

The Nose, Paranasal Sinuses, and Ears in Childhood. Donald F. Proctor. 187 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1963. $13.75.

Operating Theatre Technique. Raymond J. Brigden, S.R.N. 580 pp. Illust. E. & S. Livingstone Ltd., Edinburgh & London; The Macmillan Company of Canada Limited, Toronto, 1962. $13.50.

Ophthalmology in the Tropics. E. J. Somerset. 171 pp. Illust. Baillière, Tindall and Cox, London; The Macmillan Company of Canada Limited, Toronto, 1962. $7.20.

Pediatric Surgery. 2nd ed. Orvar Swenson. 780 pp. Illust. Appleton-Century-Crofts, New York, 1962. $20.00.

Photographic Illustration for Medical Writing. Donald J. Currie and Arthur Smialowski. With a foreword by D. C. Graham. 116 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1962. $8.25.

Post Storm King Dikes in the Hudson Highlands of New York. Seymour Mack. Annals of the New York Academy of Sciences. Vol. 93, Art. 24, pages 923-934. Editor: Harold E. Whipple. New York Academy of Sciences, New York, October 25, 1962.

Preliminary Studies on Syncoelous Parabiosis in Adult Newts, *Diemictylus viridescens*. Sophie Jakowska. Annals of the New York Academy of Sciences. Vol. 93, Art. 22, pages 877-902. Illust. Editor: Mary C. Johnstone. New York Academy of Sciences, New York, October 23, 1962.

A Primer of Pathology. F. A. Ward. 116 pp. Butterworth & Co. (Canada) Ltd., Toronto, 1962. $3.50.

Principles of Gynaecology. 2nd ed. T.N.A. Jeffcoate. 852 pp. Illust. Butterworth & Co. (Canada) Ltd., Toronto, 1962. $23.75.

Principles of the Treatment of Trauma. Basic Principles, Plastic and Orthopaedic Aspects of Trauma. Edited by W. H. Kirkaldy-Willis and A. Michael Wood. Forewords by W. Rainsford Mowlem and H. J. Seddon. 244 pp. Illust. E. & S. Livingstone Ltd., Edinburgh & London; The Macmillan Company of Canada Limited, Toronto, 1962. $7.55.

Pye's Surgical Handicraft. 18th ed. Vol. II (Special). Edited by Hamilton Bailey with the assistance of James Kyle. Foreword by the Rt. Hon. Lord Cohen of Birkenhead. 424 pp. Illust. John Wright & Sons Ltd., Bristol; The Macmillan Company of Canada Limited, Toronto, 1962. $7.20.

Recent Advances in the Measurement of Anxiety, Neuroticism, and the Psychotic Syndromes. Raymond Cattell, Ivan H. Scheier and Maurice Lorr. Annals of the New York Academy of Sciences. Vol. 93, Art. 20, pages 813-856. Illust. Editor: Mary C. Johnstone. New York Academy of Sciences, New York, October 10, 1962.

The Relatives of Man: Modern Studies of the Relation of the Evolution of Nonhuman Primates to Human Evolution. Consulting Editor: J. Buettner-Janusch. Annals of the New York Academy of Sciences. Vol. 102, Art. 2, pages 181-514. Illust. Editor: Harold E. Whipple. New York Academy of Sciences, New York, December 28, 1962.

Strontium-90 in the Soils of the New York City Area. J. L. Kulp, A. Kaufman, R. Slakter Hirshman and A. R. Schulbert. Annals of the New York Academy of Sciences, Vol. 93, Art. 18, pages 761-774. Illust. Editor: Mary C. Johnstone. New York Academy of Sciences, New York, September 28, 1962.

Surgery of Childhood. Edited by James J. Mason Brown. 1302 pp. Illust. Edward Arnold (Publishers) Ltd., London; The Macmillan Company of Canada Limited, Toronto, 1962. $36.00.

Surgical Practice of the Lahey Clinic. 3rd ed. By Members of the Staff of the Lahey Clinic, Boston. 872 pp. Illust. W. B. Saunders Company, Philadelphia; McAinsh & Co. Ltd., Toronto and Vancouver, 1962. $18.40.

Trauma to Nerves in Limbs. James E. Bateman. 453 pp. Illust. W. B. Saunders Company, Philadelphia; McAinsh & Co. Ltd., Toronto and Vancouver, 1962. $15.15.

Tumor Immunity. Conference Co-chairmen: D. B. Amos and C. A. Stetson, Jr. Annals of the New York Academy of Sciences. Vol. 101, Art. 1, pages 1-326. Illust. New York Academy of Sciences, New York, November 20, 1962.

Vascular Surgery. John B. Kinmonth, Charles G. Rob and Fiorindo A. Simeone. 501 pp. Illust. Edward Arnold (Publishers) Ltd., London; The Macmillan Company of Canada Limited, Toronto, 1962. $18.00.